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DIVERTICULITIS OF THE COLON

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This study consists of a review of the cases of patients suffering from diverticulitis of the colon who were treated at the Confederate Memorial Medical Center during the period of 1952 to 1956. An attempt has been made to review the complications of diverticulosis of the colon and not to determine its incidence. This makes comparative statistics more complex, because most of the series of reported cases compare the incidence of complicated diverticulosis and the incidence of surgery with the over-all occurrence of diverticulosis. It has been stated that approximately one out of eight barium enemas will show the presence of diverticula. The term "complicated diverticula" refers to any condition ranging from inflammation to complete stenosing diverticulitis. During the period of this review there were 28 patients who were diagnosed as having diverticulitis. The diagnoses were arrived at clinically with the aid of barium enemas, or established at the time of surgery. Of these patients, 22.2 per cent were subjected to surgery. Symptoms and signs in order of frequency were: pain, rectal bleeding, nausea and vomiting, abdominal mass and diarrhea. This is consistent with the symptoms recorded in Friesen and Schmidt's[1] series from the University of Wisconsin.

Spriggs and Marxer,[2] with a large series of patients, based their diagnoses on more nonspecific complaints. In this group, 23 patients complained of abdominal pain, 12 of rectal bleeding, 9 of nausea and vomiting, 6 of abdominal mass, and 6 of diarrhea. Diverticulitis is a disease of the older age group. There was 1 patient in his twenties, 1 in his thirties, 4 in their forties, 4 in their fifties, 7 in their sixties, 9 in their seventies, and 5 in their eighties. Of these patients, 68 per cent were females and 32 per cent males. Diverticula were present in the sigmoid colons of all but 2 patients. They were concomitantly present in the descending colon in 12 patients, in the ascending colon in 8 patients, and the transverse colon in 7 patients. Twelve patients complained of rectal bleeding ranging from bright red to tarry stools. The duration of bleeding varied from one day to several years (intermittently). Three patients required transfusions, but none were operated on for bleeding *per se*. This lends some support to other writers who contend that bleeding from diverticulitis usually ceases without surgical intervention.

Six patients in this series had operative procedures. Three of these had an inflammatory reaction in the sigmoid and descending colon, and 3 had an involvment of the cecum and ascending colon. It is interesting to note that none of these patients were definitely diagnosed preoperatively, although the diagnosis was suspected. The preoperative diagnosis was ruptured appendix in 2 patients and possible carcinoma of the ascending colon or sigmoid in the others. There is no uniformity of opinion as to the surgical procedure of choice in complicated diverticulitis. Each patient must be treated as an individual. This is well illustrated in our 6 operative cases.

CASE RECORDS

Case 1. Rectosigmoid obstruction with an inflammatory mass which required a three-stage procedure. Result good.

Case 2. Abscess and inflammatory induration of the cecum and ascending colon. Two-stage Mikulicz's procedure. Result good.

Case 3. Large mass in rectosigmoid. Primary resection.

Case 4. Perforated cecal diverticulum with an inflammatory mass in the region of the ascending colon. Ileotransverse colostomy with a good result.

Case 5. Severe diverticulitis of ascending colon with abscess. Cecostomy and right colon resection. Two stages. Postoperative course very stormy, but final result good.

Case 6. Ruptured sigmoid diverticulum with peritonitis. Exteriorization of sigmoid and later resection. Result good.

There was no mortality in this study directly related to diverticulitis.

SUMMARY AND CONCLUSIONS

There were 28 patients with diverticulitis at the Confederate Memorial Center over a 5-year period. A casual comparison was made with other reported series. The incidence appears low when considering that there are 2500 surgical admissions a year. The cases of patients subjected to surgery were reviewed and no trend to a specific procedure was noted; therefore, it is concluded that individualization of each patient is important. No early or "prophylactic resections" of the sigmoid were done for diverticulitis, nor were any patients subjected to surgery because of bleeding.

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MESENTERIC THROMBOSIS COMPLICATING OCCLUSIVE DISEASE OF THE ABDOMINAL AORTA: CASE REPORT*

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Acquired diseases of the aorta, occlusions and aneurysms, have been more widely recognized in the past few years. As specific occlusions of major vessels are diagnosed with increasing accuracy and frequency, the error of classifying these cases as Buerger's Disease or simply "generalized arteriosclerosis" is made less often. The methods for more specific diagnoses (detailed history, complete physical examination, arteriography) have been available for years, but only recently have they begun to be adequately utilized. Too often competent doctors take the easy road for diagnosing these cases, *i.e.*, attach a name, prescribe a drug, of questionable value, or do a sympathectomy, and complacently feel that all has been done. However, all has not been done, until the correct specific diagnosis has been made and proper therapy recommended.

There has been a great advance in the treatment of aneurysmal and occlusive disease since it was learned that excisional and replacement therapy was feasible. Authors interested in this work have stressed early treatment of chronic, insidious occlusion of the abdominal aorta[6] before propagation of the thrombotic process occludes the renal or superior mesenteric arteries. Reports of death due to this complication are rare.[4] For this reason we are reporting such a case—a patient whose disease was mistaken for Buerger's Disease for years.

REPORT OF CASE

D. L., a 55-year-old retired steel mill worker, was admitted to the St. Luke's Hospital, Jacksonville, Florida, on August 31, 1956 complaining of severe epigastric pain of 15-hr. duration. There was a history of several similar transient episodes. There had been no pain between attacks. The pain was localized to the abdomen without radiation. Vomiting occurred with the last two attacks, but there was no hematemesis. The admission diagnosis was coronary thrombosis.

The patient noted the onset of easy fatigability in both legs in 1942, which thereafter grew progressively worse. In 1943, he developed intermittant claudication in both calves which also became slowly progressive. At the same time, he noted numbness in both hips associated with the claudication and fatigue. His local physician in Pennsylvania diagnosed his difficulty as Buerger's Disease. He was referred to a specialist who concurred with the diagnosis. The patient took two courses of hydrotherapy at Hot Springs, Arkansas, with only temporary benefit. In the same year he was no longer able to continue his work. In 1945, he consulted a clinic in Cleveland, where the diagnosis of Buerger's Disease was retained. Typhoid fever therapy and deep whirlpool therapy were given without noticeable effect. There had been a known absence of both femoral and pedal pulses for years. The patient denied noticeable alteration in his sexual libido. He had been a heavy smoker all of his adult life.

The patient had been a known hypertensive for at least 15 years. In April 1953, he had a coronary occlusion with associated congestive failure. He experienced no subsequent occlusion, although he had mild exertional dyspnea. In October 1953, he suffered a mild cerebrovascular accident from which he made an early recovery.

The patient and his family moved to Jacksonville, Florida in 1954. In July 1955, he was found to have a large, nodular, toxic goiter. This was treated with radioactive iodine by Dr. Samuel Root. Several residual nodules increased in size and in July 1956 the senior author (S.M.D.) performed a total thyroidectomy. Recovery was uneventful. In the course of examination it was recognized that the patient's vascular disease was probably the result of arteriosclerotic occlusive process proximal to the femoral arteries. The significance of these findings was not appreciated. It was thought that the patient could live with his disease. He curtailed his activity so that he was reasonably comfortable. No definitive therapy was advised. That this reasoning was in error was shown by his subsequent course.

When admitted to the hospital in August 31, 1956, his acute pain had subsided. His skin was pale, cool and clammy. His blood pressure normally was 180/90; at the time of admission it was

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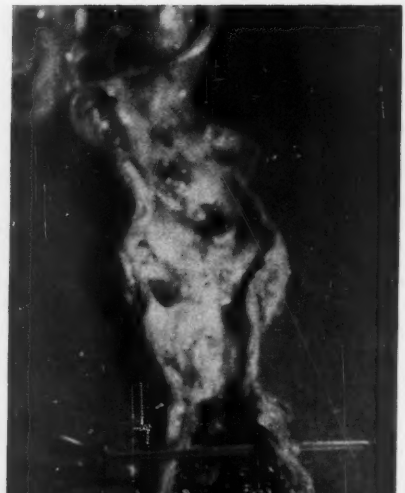

FIG. 1. The aorta has been sectioned on its anterior surface demonstrating the aneurysmal and occlusive disease. A probe has been inserted into the left renal artery, which contained a short extension of the proximal fresh premortem thrombus. The superior mesenteric artery is not demonstrated.

110/90 with a pulse of 132. His temperature was 99° F. and there was a normal sinus rhythm. The heart was not grossly enlarged. There was a grade I apical systolic murmur. The abdomen was slightly distended and nontender. No masses were palpable. The liver and spleen were not enlarged. Peristalsis was present. The femoral and pedal pulses were absent bilaterally. The legs were slightly pale.

The red blood cell count was 5,100,000, and the hemoglobin 16.7 gm. per cent. The electrocardiogram indicated myocardial damage, probably old. A chest roentgenogram revealed slight left ventricular enlargement; an enlargement of the left hilum thought to be an aneurysm of the distal arch, and two dilatations of the descending thoracic aorta. Abdominal roentgenograms revealed a small amount of gas in the small intestine.

Diagnostic impressions were (1) dissecting aneurysm, (2) coronary thrombosis, (3) mesenteric thrombosis, and (4) pancreatitis.

Shortly after admission there was a recurrence of abdominal pain which persisted until death. The severity of pain increased. On September 1, the day after admission, his abdomen became markedly distended. Repeat roentgenograms revealed marked gastric dilatation which was suggestive of paralytic ileus. Urinary output was diminished. There was moderate lower abdominal tenderness, midabdominal tympany, and flank dullness. Urinalysis showed 4 plus albumin, 35 to 40 white blood cells and no red blood cells. The serum amylase had risen to 500 mg. per cent, the leukocyte count was 27,500, and the hemoglobin was 17.2 gm. per cent.

The authors saw the patient in consultation 9 hr. before death. Acute hemorrhagic pancreatitis was the first impression, although the possibility of mesenteric thrombosis was discussed. The mechanism of mesenteric thrombosis complicating advanced arterial occlusive disease was not considered. Here we were in error because the inherent dangers of the disease were not realized. Flank paracentesis recovered a small amount of brownish serosanguineous fluid, which was insufficient for amylase determination. The patient's condition was too poor to consider surgical exploration. Blood and norepinephrine were administered without appreciable benefit until death. Permission for autopsy was obtained.

At autopsy, the thymus was present and enlarged, measuring 6 by 3 by 2 cm. The heart weighed 700 gm. and showed diffuse moderate hyperthrophy, more prominent of the left ventricle.

The abdomen was moderately distended. The abdominal cavity contained 100 cc. of brown hemorrhagic fluid. The liver and spleen microscopically showed chronic passive congestion. The pancreas was lobulated and microscopically appeared normal. The stomach and small bowel contained thin hemorrhagic fluid. There was hemorrhagic infarction of the entire jejunum and ileum. There was no microscopic evidence of infarction of either kidney, although moderate degenerative changes compatible with the patient's age and his arterial disease were demonstrable.

The aorta showed severe, irregular, diffuse atherosclerosis. The thoracic aorta contained three aneurysms, including a fusiform aneurysm of the descending arch distal to the left subclavian artery. There was a wide saccular aneurysm of the distal abdominal aorta. The lumen of the abdominal aorta was completely occluded by dense, yellow fibrous material from a level just below the renal arteries to the external iliac arteries bilater-

ally. Both external iliac arteries were of average size, but had moderate arteriosclerotic changes. The lumen of each was diminished. Proximally, a fresh thrombus incompletely occupied the lumen of the aorta so as to overlay the lumen of both the left renal and superior mesenteric arteries. The lumen of the latter vessel contained no thrombus, while the left renal artery contained a fresh loosely attached thrombus (fig. 1). Microscopic sections through the occluded aorta and common iliac vessels revealed a dense laminated hyaline material. The walls showed prominent medial atrophy, scarring, chronic inflammation, cholesterol crystal deposits, and deposits of amorphous debris. There was a dense periaortic fibrous tissue reaction involving contiguous structures.

DISCUSSION

The clinical pattern of this patient's disease conforms to the original description given by Leriche[6] in 1923, but not published in English until 1948.[7] Our patient developed his first symptoms at the age of 40 with progression thereafter. Briefly these symptoms and signs can be enumerated as follows: (1) easy fatigability of the lower extremities, (2) progressive intermittent claudication, (3) numbness or pain in the hips and back, (4) minimal color and trophic changes in both lower extremities, (5) absence of pulsations in large and small arteries of the lower extremities, (6) hypertension, (7) and the presence of a midabdominal bruit. Only one significant complaint was lacking—that of loss of sexual libido.

It is not surprising that the stigma of Buerger's Disease was applied to this patient 15 years ago. This error was understandable at that time. The true clinical picture was not completely recognized until postmortem examination. This fact clearly demonstrates the need for earlier diagnosis in many such patients. The reporting of complications that may arise from delays of therapy will emphasize the need for correct diagnosis and early treatment.

As seen in figure 1, the occlusive process in this patient was complete. DeBakey and associates[1, 2] have classified the disease process into two types, those with complete occlusion of the aortic bifurcation, and those with partial occlusion. Some elevation of the blood pressure is present in the great majority of patients with both types of disease. This is thought to be chiefly the result of the increased resistance to flow at the site of obstruction. Abdominal and femoral arteriography may be necessary to correctly identify the extent of disease.

While the fundamental process is the same in each, there are certain distinguishing characteristics which are of value in diagnosis, treatment, and prognosis. In complete occlusion, the average patient is younger, the pathologic process is more localized, and the results of treatment are more favorable. It is uncommon to find occlusion of the external iliac and femoral arteries due to arteriosclerosis in this group, thus permitting more effective linear flow with the use of a bifurcation aortic graft.

In the incomplete type of occlusion, patients are in an older age group, and peripheral vessels, as well as the aorta, are more diffusely involved. In the majority of such patients there is complete occlusion of one or the other common iliac arteries. In these patients, successful treatment depends, not only upon the use of a bifurcation graft in most instances, but frequently the use of additional ilio-femoral grafts as well. Extensive endarterectomy to obviate use of such peripheral grafts has been distinctly unsuccessful, due to postoperative thromboses.

The patient described herein was unusual because of the presence of both major types of acquired aortic diseases, aneurysmal, as well as, occlusive. As seen in figure 1, there were separate and distinct aneurysms of the thoracic aorta—one sacciform and two fusiform. These were diagnosed by a competent roentgenologist as a prominent left pulmonary artery. Probably this diagnosis was made because he was not as "aneurysm conscious" as one should be today. Another interesting feature is that while dilatation of the occluded segment of the abdominal aorta in the Leriche syndrome is rare,[1] it was present in this instance in an unusual degree. Also, contrary to statements made above concerning complete occlusive disease, the external iliac arteries were extensively diseased, although there remained a small lumen in each.

As stated repeatedly by other authors,[1-5] the rationale for advising early treatment of occlusive disease of the aorta is that the thrombotic process will be propagated upward and/or downward as the disease progresses, and one can eventually expect occlusion of the renal, superior mesenteric or femoral arteries to occur. The proximal thrombus is soft, dark, and loosely attached because of its more recent origin. Occlusion of the orifice of

the left renal and superior mesenteric arteries was clearly demonstrated at postmortem examination in the patient described, re-emphasizing a serious consequence of this disease.

SUMMARY

A patient with chronic occlusive disease of the terminal abdominal aorta is presented. Because corrective therapy was not advised before the terminal episode, the patient died from mesenteric occlusion due to ascending thrombosis of the aorta complicating the disease.

Patients with absent femoral pulses and with symptoms of occlusive disease of the abdominal aorta should be studied thoroughly. Those with a diagnosis of Buerger's Disease, or "arteriosclerosis" without a localized occlusion, should be re-evaluated. Differentiation from other types of vascular disease should be made and arterial occlusions specifically localized.

Patients presenting persistent symptoms, suggesting unexplained urologic, orthopedic, neurologic or general surgical conditions of the abdomen, back or lower extremities, should be given the benefit of a thorough vascular study.

Even though it may be said, "The patient has learned to live with his disease, let him alone," serious complications may develop. The importance of correct, early diagnosis and appropriate treatment is stressed. It is only through such efforts that serious complications of this disease can be avoided.

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RETROPERITONEAL CHYLOUS EFFUSION: A CASE REPORT

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Effusion of chyle into the retroperitoneal space associated with alarming symptoms must be very rare, judging by a review of the English literature. Chylothorax and chylous ascites are effusions of somewhat similar nature and are well documented.[1-3] The specific etiology of these entities is varied and often unknown, but such effusion is generally thought to be the result of lymphatic obstruction and is usually associated with neoplasm of the stomach, with retroperitoneal and mediastinal lymph node enlargement, by neoplasm or tuberculosis, or with trauma. More rare conditions have been described, including lymphatic obstruction with chylous ascites due to irradiation reaction following x-ray therapy to the left renal area after removal of a Wilm's tumor.[4] Retroperitoneal effusion probably has a similar mechanism. Because this particular entity has not been described and because of the interest in any condition simulating "the acute abdomen," the following case report is presented.

CASE REPORT

History: C. M., a 32-year-old Negro man, entered the hospital with the chief complaint of abdominal pain of two days duration. He was in the army and was treated for jaundice in 1942. He was discharged in 1943 with the diagnosis of psychoneurosis—anxiety hysteria. Since that time he has frequently had mild lower abdominal pains associated with nervous spells. These attacks were mild and never necessitated his losing time from his work as a mail carrier. The present illness started two days prior to admission as one of his usual attacks of mild lower abdominal pain. However, the pain was more persistent and became a girdle-like pain, radiating from the umbilicus around both sides to the back. He then noticed that his abdomen became distended and "full of gas." Six to eight hours later the distention disappeared and he felt better. During the day prior to admission the pain became progressively more severe, and became most severe in the right lower quadrant. There was no history of nausea, vomiting, diarrhea, constipation, weight loss, fever, chills, or urinary symptoms. There was no unusual ingestion of fat.

On physical examination the temperature was 98.6°F.; the pulse rate was 80 per min.; the respiratory rate was 20 per min.; the blood pressure was 120/80. The patient was well nourished and well developed, appearing mildly, acutely ill, lying in the supine position with his legs drawn up, and holding his hand over his right lower abdomen. General physical examination was negative except for the following: The abdomen was slightly distended. There was moderate tenderness over the entire abdomen, most marked in the right lower quadrant. There was mild rebound tenderness over the abdomen most marked in the right lower quadrant; there was no referred tenderness. The bowel sounds were hypoactive but were of normal quality. No organs or masses were palpable. Rectal examination revealed moderate tenderness at the tip of examining finger.

Laboratory findings. The white blood count on admission was 8,700 cells per cu. mm. with 67 per cent neutrophils, and 33 per cent monocytes. Two hours later the white blood count was 10,200 cells per cu. mm. The hemoglobin was 15.0 gm. per 100 cc. Urinalysis revealed clear amber urine with specific gravity 1.015, pH 5.0 and negative test for sugar and albumin; the urine sediment was normal. Blood chemistry studies were as follows: Venereal Disease Research Laboratory was negative; serum amylase 90 diastase units (up to 200 units normal); serum lipase 2.1 units (normal 0.5 to 1.0 units). Thymol flocculation negative; Zn SO_4 turbidity 5.5 units; thymol turbidity 2.0 units; alkaline phosphatase 1.3 units; cephalin flocculation negative; bilirubin 0.08 units direct and 0.16 units indirect; total lipids 562 mg. per 100 cc. Radiologic studies showed on admission a normal chest x-ray and normal flat and erect x-rays of the abdomen. Postoperative x-ray studies including barium esophagram, upper gastrointestinal series, barium enema, intravenous pyelograms, and oral cholecystogram were normal.

Operation. After pentothal induction nitrous oxide-ether-oxygen anesthesia was administered.

A right lower quadrant muscle splitting incision was made. As the properitoneal space was entered, a cloudy, thin, odorless, white fluid, looking like milk, exuded into the wound. Further dissection revealed that this fluid was located immediately under the peritoneum, and throughout the remainder of the procedure this milky fluid continued to enter the wound. The peritoneum was opened and several bands between the cecum and lateral parietal peritoneum were divided. Exploration revealed extensive subserosal infiltration by the milky fluid involving the posterior parietal peritoneum, the cecum and ascending colon, and the base of the small bowel mesentery. The lacteals in the small bowel mesentery were distended and visualized as white streaks, but there was extravasation of this material only in the base of the mesentery. The appendix appeared normal; it was removed in routine fashion and the stump inverted. During the operation, approximately 150 cc. of fluid accumulated in the pelvis. It was aspirated and saved for laboratory analysis. It was thought at this point that the patient had a chylous effusion and that nothing would be gained by more extensive exploration through another incision. The wound was closed in layers. A small rubber drain extending to the peritoneum was brought out through the incision.

Pathology report on the appendix: Normal. Examination of the chyle revealed milky white fluid, with a specific gravity of 1.013. Chemical analysis revealed the following: sodium 5 mEq. L. per 100 c.; potassium 1.2 mEq. per. L ; glucose 10.3 mg. per 100 cc.; chlorides 14 mEq. per L.; calcium 4.8 mg. per 100 cc.; inorganic phosphorus 1.03 mg. per 100 cc.; total proteins 2.2 gm. per 100 cc.; albumin 1.6 gm. per 100 cc.; globulin 0.6 gm. per 100 cc.; cholesterol total 51.5 mg. per 100 cc.; cholesterol esters 45.5 mg. per 100 cc. (88 per cent); phospholipids 290 mg. per 100 cc.; total lipids 1630 mg. per 100 cc. Bacteriologic examination of the chyle showed no bacteria on smear; culture revealed diphtheroids and hemolytic staphylococcus albus sensitive to aureomycin, achromycin, bacitracin, penicillin, chloromycetin and terramycin, and resistant to dehydrostreptomycin.

Hospital course. The patient's postoperative course was benign. His temperature fell from 101.8°F. immediately after the operation to normal on the third postoperative day. Gastric suction had been instituted prophylactically and was discontinued on the second postoperative day. The wound drained only a minimal amount, and the drain was removed on the day after operation. The wound healed *per primum*. The temperature elevation in the postoperative period in the presence of an unusual and poorly explained entity encouraged the surgeon to give the patient aqueous penicillin and dihydrostreptomycin for five days. These drugs did not appear to affect the course of recovery. The patient was asymptomatic following operation and after completing the diagnostic studies he was discharged on the thirteenth hospital day. Six months after the operation. the patient is well, working, and symptom free.

DISCUSSION

The diagnosis of retroperitoneal chylous effusion would appear to be very difficult to make preoperatively. No diagnostic features were demonstrable in this case, except perhaps the elevated serum lipase. However, the operative findings are striking, and if this condition is encountered during laparotomy, simple closure of the wound without drainage would seem to be the procedure of choice, assuming that no other pathologic condition exists which needs definitive treatment. Complete x-ray and laboratory studies failed to reveal other diseases such as lymphoma, tuberculosis, etc. The conclusion is rather reluctantly drawn that spontaneous rupture of a lymphatic channel or a retroperitoneal chylous cyst[5] occurred.

SUMMARY

A unique case of idiopathic chylous effusion simulating "the acute abdomen" is presented.

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AN EVALUATION OF CYSTIC TUMORS OF THE NECK*

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Cystic tumors of the neck are not uncommon. Although usually seen in adults, the majority are congenital in origin and may be traced to embryonic structures. The great majority of such cystic lesions are benign, but malignant changes in the lining epithelium may be seen occasionally. Some of these tumors are difficult to eradicate surgically because of their intimate relationship to important structures.

Cystic tumors of the neck include:

1. Epidermoid or sebaceous cysts.
2. Branchial cysts.
3. Lymphatic cysts or cystic hygroma.
4. Thyroglossal duct cysts.
5. Cystic papillary lymphomatosum of the parotid gland.
6. Cysts with malignant changes. (Found in epidermoid and sebaceous cysts, rarely in branchial cleft cysts and, occasionally, in thyroglossal duct cysts from aberrant thyroid tissue in the cyst wall).
7. Cystic degeneration of lymph nodes involved by metastatic carcinoma, lymphoma or inflammation.

Conditions related to trauma such as rare air cysts, esophageal diverticular sacs and acute abscess formations are not included in the discussion.

EPIDERMOID OR SEBACEOUS CYSTS OF THE NECK

Varying modes of origin have been confirmed for these cysts. Many appear to be the result of embryonic inclusions and a definite familial history may be obtained occasionally from patients. Some of the dermoid cysts found in the lateral neck arise from the precervical sinus formed from the branchial grooves. Others are retention cysts produced by occlusion of sebaceous glands or hair follicles, and some arise by traumatic displacement of epithelium.

Benecke's classification, adapted from Chiari, is in agreement with the views of the authors in a study of 400 cases, namely: "follicle cysts" are true retention cysts of sebaceous glands or hair follicles (fig. 1 *A* and *B*), and "epidermoid cysts" (dermoid cysts) represent embryonic inclusions (fig. 2). The character of the cyst lining will depend upon the state of differentiation of the misplaced epithelium. Undifferentiated primitive basal cells displaced below the surface will differentiate in two directions: towards squamous cells, with pearl formation, and towards skin appendages. If differentiation has occurred before displacement, the cyst lining will usually contain only simple squamous cells. Follicle or epidermoid cysts are the most common cystic tumors found in the neck, although they may be found in other parts of the body. The greatest number are found in the neck of young adults. The average duration of symptoms is given in terms of years rather than in months. Some patients place the clinical onset in early childhood. The epidermoid (dermoid) cyst is usually covered by freely movable skin but is attached to the deeper fascia periosteum or cartilage (ear) over which it lies. The sebaceous (follicle) cyst is always attached to skin and moves with the skin over the deep structures. The lesions vary from 1 to 5 or 6 cm. in diameter, usually situated just below the epidermis. Frequently a white, waxy substance can be expressed from one or more dilated sebaceous ducts connected with the cyst.

In 150 patients with sebaceous or epidermoid cysts of the neck we have observed only two instances of malignant change. Both cysts showed squamous cell carcinoma. Ten such malignant cysts were found in epidermoid cysts elsewhere in the body.

Treatment. All sebaceous and epidermoid cysts should be removed because of growth potential and the definite incidence of malignant change noted in these lesions. It is best to remove them by complete excision. If the cyst is infected or if there is abscess formation, the treatment should be limited to incision and drainage or the use of hot compresses. In the presence of infection, the

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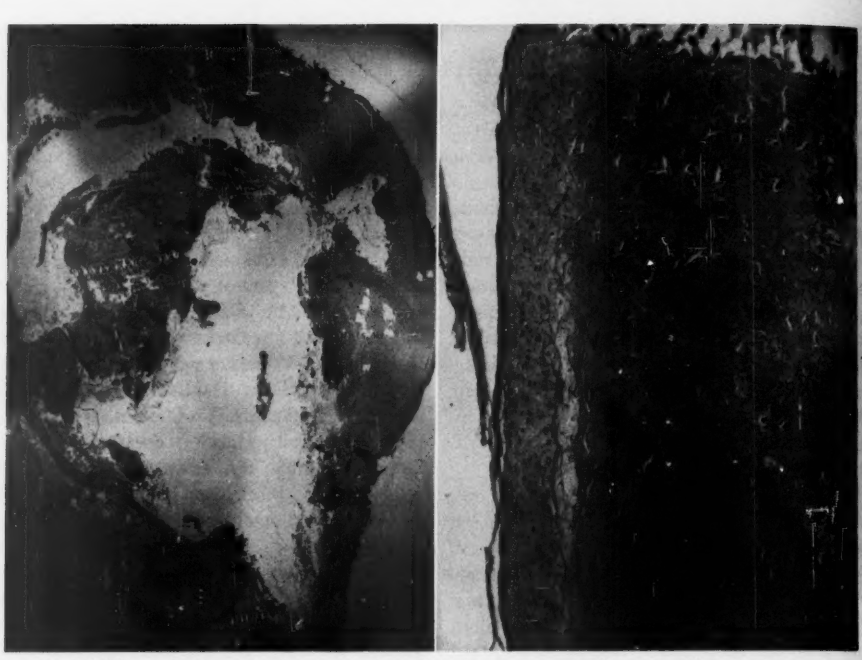


FIG. 1. (*A*) Microphotograph of cross section of a sebaceous cyst. Note the debris in the cyst cavity with extensive deposits about the cyst wall. (*B*) Microphotograph showing cyst wall with compression of lining cells by sebaceous debris.

removal of such a cyst lining should be deferred until the infection has subsided.

In removing epidermoid cysts about the ear it is often better to do the operative procedure under a general anesthesia because of the frequent ramifications of the cyst at its base. When an epidermoid cyst is situated in the pre- or post-auricular region it may extend along the ear cartilage to the skull region. This may require ablation of the residual cyst lining by chemical cauterization (using phenol followed by 95 per cent alcohol).

BRANCHIAL CYSTS AND FISTULAS

Imperfect obliteration of the branchial "clefts" (reminiscent of the gill-slit condition in aquatic vertebrates) leads to the formation of cervical branchial cysts or fistulas. The second cleft, cervical sinus and third pharyngeal pouch have been cited as sources of origin for such anomalies.

We believe that the complex arrangement of cervical grooves and sinuses forming opposite the third branchial pouch which opens on the anterior border of the sternomastoid muscle is concerned with the development of branchial cysts after birth (fig. 3). The cervical sinus tract extending upward toward the tonsil is responsible for the branchial fistulas reaching the tonsillar pillar, and the cervical vesicle for the majority of branchial cysts (fig. 4). The embryonic relation of the cervical vesicle and sinus to the tonsil and thymus is apparently responsible for the association of lymphoid tissue with branchiogenic tumors and cysts.

We have studied 55 patients with benign branchiogenic cysts and fistulas. The majority of these lesions were found in adults. In a few instances the tumor was noted at birth. The duration of swelling is often noted as being present for from 5 to 10 years with gradual increase in size. Upper respiratory infection often precipitates a rapid increase in size of the cystic swelling, associated with infection of the cyst.

In the typical case, a soft or fluctuant tumor from 3 to 6 cm. in diameter is found below the angle of the jaw, anterior to the sternocleidomastoid muscle. Cysts often have been confused with inflammatory lymph nodes, clinically, and frequently may be infected at operation.

Rarely does the cyst communicate with the

FIG. 2. Epidermal inclusion cyst with squamous cells lining the cavity. There was no sinus leading to surface of skin.

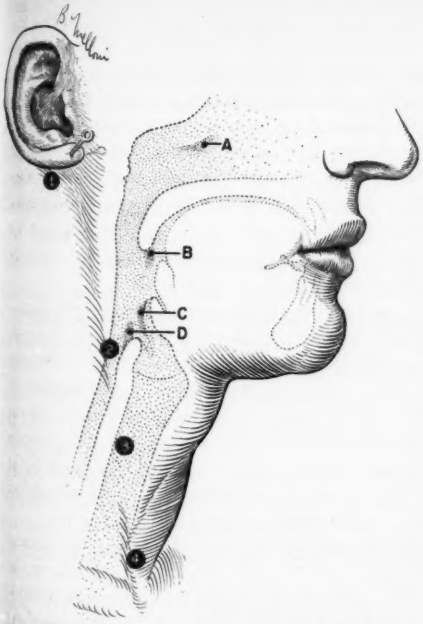


FIG. 3. Composite diagram of adult head to indicate the sites of the embryonic pouches (A, B, C, D) and preferred locations of cervical cysts, diverticulas and fistulas (1, 2, 3, 4).

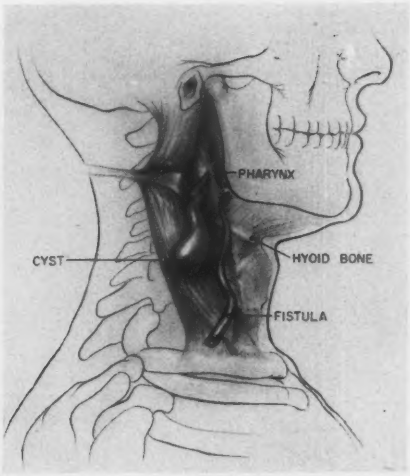


FIG. 4. Composite drawing to illustrate (a) cervical cyst derived from an ectodermal groove or a complementary entodermal pouch, (b) cervical fistula, an open communication between pharynx and external surface of neck due to incomplete closure of branchial cleft.

Cervical diverticulas are incomplete fistulas leading outward from the pharynx or inward from the skin of neck appearing in the same locations. The external openings of diverticulas or fistulas tend to be drawn down to levels lower than the site of origin.

exterior skin surface by fistula. On the other hand a fistulous tract may be found coursing through the deep structures of the neck beginning at various levels above the supraclavicular region and extending up to the tonsillar fossa, without evidence of cyst formation.

Branchial cyst walls may vary in thickness and the cavity may contain either a white cheese-like material or greenish to yellow liquid (fig. 5). The cyst wall, microscopically, shows transitional epithelium overlying lymphoid tissue with germinal centers (fig. 6 *A* and *B*). Occasionally the epithelial lining is absent because of infection. The fistulous tracts may show cuboid or columnar epithelium near the pharynx portion of the tract. A branchial appendage may be found accompanying a sinus tract characteristically near the level of the cricoid cartilage or just beneath the lobe of the ear.

Treatment. Branchial cysts should be completely excised, preferably under general anesthesia. Repeated aspirations, incision and drainage (except with acute infection), sclerosing agents or x-ray treatment are not recommended for definitive treatment.

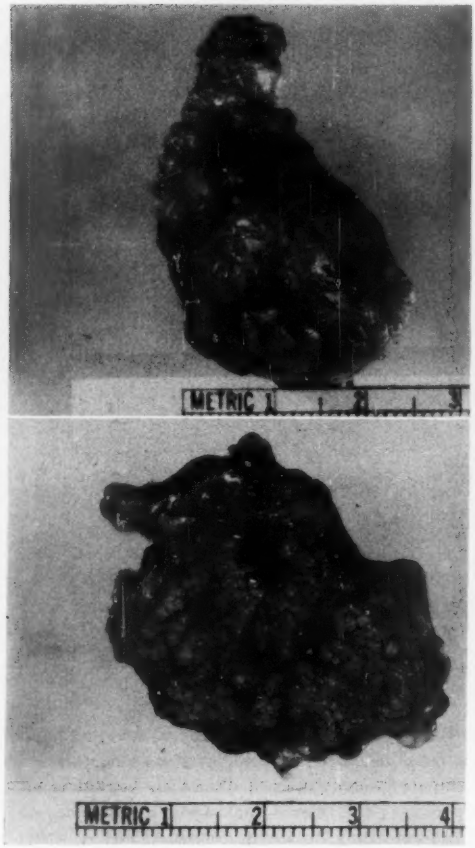


FIG. 5. Branchial cyst which in cross section shows wall with many rugae and much enfolding of mucous membrane. Marked submucosal lymphoid hyperplasia increases thickness of the wall.

An adequate exposure is made by incising along the anterior border of the sternocleidomastoid muscle directly over the cyst (fig. 7). The cyst wall is identified and freed from its bed by careful, blunt and sharp dissection (fig. 8). Care should be exercised not to rupture the cyst and any prolongation of the cyst must be dissected out to insure complete removal. Extremely large cysts may require evacuating the contents before dissection is begun. The wound is closed in a routine manner at the discretion of the operator. It is well to insert a small rubber drain for two or three days.

In treating fistulous tracts, the extent of the tract should be determined preoperatively, if possible. An opaque substance such as lipiodol is injected into the tract through a ureteral catheter or an appropriate blunt needle on a syringe. It is best to use local anesthesia about the orifice of the fistula before injection. A subsequent roentgenogram will usually reveal the course of the tract.

Although sclerosing agents have been used to obliterate the tract, we believe that excision is by far the best method of treatment. The Hamilton Bailey procedure has proved the most satisfactory method for surgical resection. Preferably under general anesthesia, a purse-string suture is placed about the fistulous opening, firmly closing the tract. An elliptical incision is made about the orifice. The tract is dissected out (usually upward) by means of blunt and sharp scissors dissection. If necessary a second transverse incision is made at the appropriate level to continue the dissection of the tract. If the tract extends to the pharynx, intrapharyngeal pressure by an assistant's finger placed through the oral cavity will enhance the ease with which the tract may be followed by dissection into the pharynx. The pharyngeal defect is closed with interrupted, inverting fine chromic catgut sutures in the mucosa, and fine interrupted silk sutures in the submucosal tissues. The wounds are closed in a routine manner with interrupted sutures of fine silk. A drain is inserted through the lower wound along the course of the tract for two or three days. This stepladder type of incision is preferred, cosmetically, to an extensive longitudinal incision along the anterior border of the sternocleidomastoid muscle.

BRANCHIOGENIC CARCINOMA

In a review of the medical literature, a small number of case reports present reasonable evidence that a carcinoma found in the neck may be of branchiogenic origin.

Von Volkmann in 1882 thought that some cervical growths arose from vestiges of the branchial grooves (not knowing the primary site of the tumors). Ewing, in 1919, reported similar cases as arising in branchogenic vestiges, although his report reflected symptomatology frequently consistent with undetected primary pharyngeal cancer.

Skeptics, such as Bland Sutton (1893), Willis (1934) and Martin and associates (1950) have challenged the existence of branchiogenic carcinoma and have called attention to the probabil-

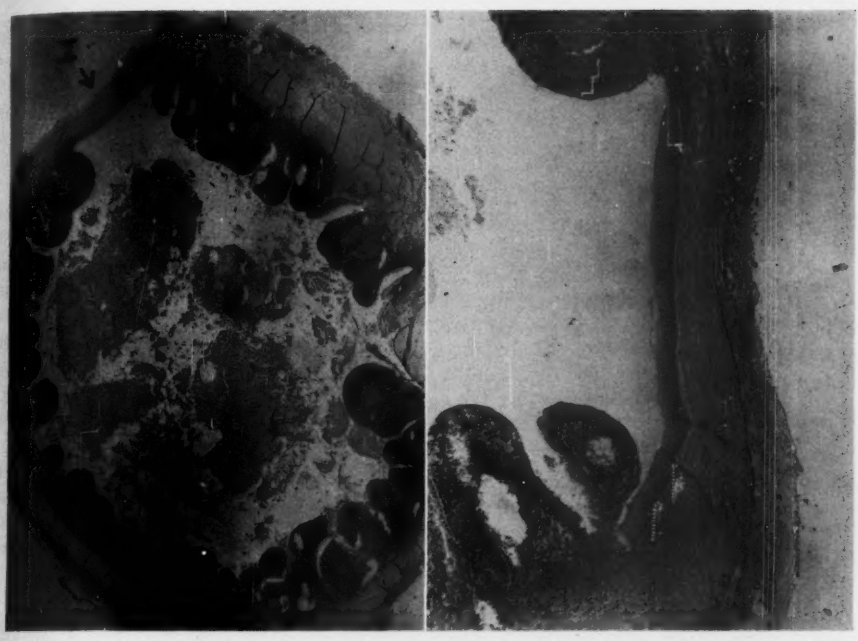


FIG. 6. (*A, left*) Microphotograph of the branchial cyst wall showing transitional cell epithelium overlying islands of lymphoid tissue with germinal centers. (*B, right*) Microphotograph, higher magnification, showing area I from figure 6 *A* indicating the absence of lymphoid tissue and with hyperplasia of transitional cell epithelium lining the wall.

ity of silent primary lesions in the mouth and pharynx. They have emphasized the necessity for careful examination of the head and neck for unsuspected primary lesions which metastasize to the lymph nodes of the cervical region.

Martin, Morfit and Ehrlich, in a report from the Memorial Hospital in 1950, could find only 15 among 5000 patients with primary tumors above the clavicle which they were willing to tentatively classify as branchiogenic carcinoma. A critical analysis of these patients and the literature led these authors to conclude: (1) that at the present time no proof is available to support belief in the existence of such a tumor; (2) that there may be no other more reasonable explanation for certain rare cervical tumors; (3) that a definite diagnosis of branchiogenic cancer cannot be made on a histologic basis; (4) that the diagnosis of branchiogenic cancer should remain tentative and should not even be considered unless the patient had passed five years without evidence of a primary tumor elsewhere.

Ward and Hendrick reported 7 instances of branchiogenic carcinoma out of 70 patients with branchiogenic anomalies. Three of these lesions were thought to have arisen from definite branchial cysts.

Branchial cysts certainly constitute a readily identifiable variety of lesions arising in the neck from embryonic branchial vestiges. Cancers found definitely arising in the identifiable cysts are rare. The demonstration of a definite branchial cyst with a cancer arising from its wall, with the absence of a primary lesion elsewhere, seems to us of paramount importance in establishing the case for branchiogenic carcinoma.

We have observed one such case in which a squamous cell carcinoma was found in the wall of the branchial cyst. A radical neck dissection showed no residual disease. The patient is well at the end of five years. Only 3 patients out of 77 (recorded in the laboratory and with follow-ups) have remained well beyond five years and met the required criteria by which it is possible to make the diagnosis of branchiogenic carcinoma.

Making the clinical diagnosis of branchiogenic carcinoma is presumptive when a discrete circumscribed or indurated mass is found in the

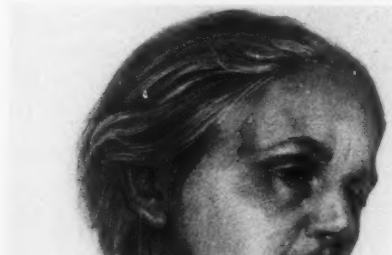


FIG. 7. The preferred incision used in removing a branchial cyst is made along the anterior border of the sternocleidomastoid muscle overlying the cyst.

upper neck, presenting anterior to the sternocleidomastoid muscle, and with or without regional lymph node enlargement. A survey of the head and neck and elsewhere for primary carcinoma is mandatory and emphasizes the necessity for a differential diagnostic evaluation of the seemingly primary tumor. Among the lesions to be considered other than branchiogenic carcinoma are: branchial cleft cyst (with or without infection); follicle or inclusion cyst; carotid body tumor; discrete lymphosarcoma; discrete Hodgkin's disease; lymphangioma; aneurysm; inflammatory lymph nodes; thyroid carcinoma with metastases to regional lymph nodes; regional metastases from a silent primary carcinoma in the parotid gland or mucous membrane-lined recesses of the head.

Having established in so far as possible the absence of a primary carcinoma which may have metastasized to the neck lymph nodes, a biopsy is in order to establish the disease as a malignant

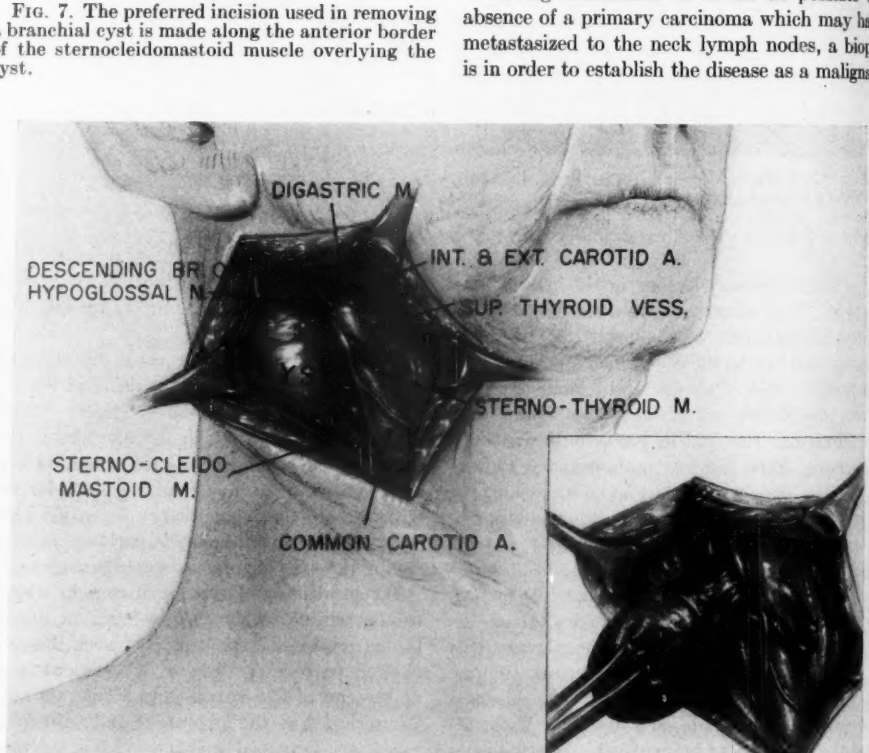


FIG. 8. The cyst wall is identified and freed from its bed by careful blunt and sharp dissection. Care should be exercised not to rupture cyst, and prolongations of cyst must be completely removed by dissecting the tract out.

process. Aspiration biopsy may be performed prior to radical surgery, or a surgical biopsy may be accomplished at the time of the projected definitive operation.

The technique of aspiration biopsy was perfected in 1934 by Martin and Ellis at the Memorial Hospital in New York. While this method has proved successful in a number of patients, it is preferable to perform a surgical biopsy, utilizing a small portion of the planned incision. The skin is opened and the tumor mass identified. If the mass is large and fixed, an adequate piece is excised and the tumor capsule closed. If the tumor area is amall, the nodule may be removed *in toto* for biopsy.

Treatment. Carcinoma having been established as the histopathologic diagnosis, and clinically evaluated as being a probable primary lesion, the surgical removal requires a radical neck dissection on the affected side. Postoperative irradiation does not seem indicated unless the operator is doubtful as to the complete ablation of the disease by surgery.

Lesions which appear to be primary and also inoperable may be palliated by treating with appropriate x-ray or telecobalt therapy. Up to 4000 roentgens delivered by the x-ray or cobalt unit may be given per field in divided dosage without undue morbidity, if the portals are not more than 10 cm. in diameter. The effect of the irradiation may produce pharyngeal and laryngeal irritation of moderate degree. Temporary improvement with some regression of the lesion may be obtained.

LYMPHATIC CYSTS OR CYSTIC HYGROMA

Cystic lesions of the neck may develop from improper development of lymphatic plexuses. The early formation of lymph spaces in the embryo appear in the region of the neck near the junction of the jugular and subclavian veins. Cyst formation may be the result of a failure of the lymphatic plexuses to unite with the veins, or they may arise on the basis of a neoplastic overgrowth of the lymphatic plexus, sometimes connected with veins.

Lymphatic cysts or hygromas are most common in young children and usually are clinically present at birth, although they may not be observed until early adulthood. We have 35 such patients on record in our files. Ninety per cent of the lesions occur in the neck, although they may be seen primarily in the axilla, retroperitoneal or popliteal areas. Their location in the neck is usually the supraclavicular region, although they may appear higher in the neck and invade the submaxillary zone and floor of the mouth. Extensive lesions are seen extending below the clavicle and also into the axilla.

There are usually no symptoms except the soft-compressible tumor, often lobulated, with progressive enlargement. Localized areas of induration may be felt within the area of compressible tumor. The skin over the tumor remains smooth but may become discolored with rupture of capillaries present. When the child cries or coughs, temporary rapid swelling or dilation of the growth occurs.

As the lymphatic cystic tumor enlarges, it tends to dissect along fascial planes, penetrate muscles and nerve sheaths, and to surround the large blood vessels. This accounts for the extensive ramifications of the disease seen in the head and neck in which the process may invade the floor of the mouth and the tongue, as well as the wall of the pharynx (fig. 9).

The disease is quite variable in its growth po-

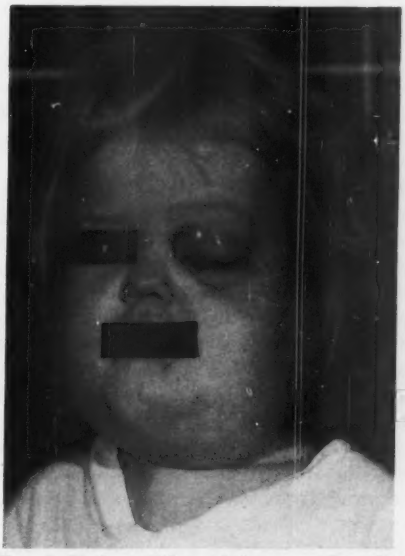


FIG. 9. Patient with cystic hygroma (lymphatic cysts) of neck, submaxillary zone, floor of mouth, tongue, pharynx and orbit. The lesion about the orbit has lymphangiomatous features.

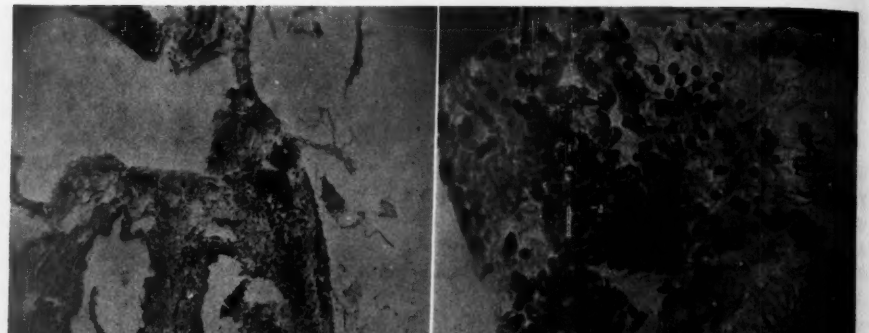


FIG. 10. (*A, left*) Note the many dilated cystic spaces lined by endothelium. Aggregates of lymphoid tissue may be seen in the interstices of the endothelial lined spaces and stroma. (*B, right*) High power microphotograph of lymphoid tissue in stroma about a lymphatic cystic space.

tential. If the lesion develops slowly it permits the surrounding structures to accommodate. If the growth is rapid, pressure symptoms may occur rapidly with embarrassed respiration, difficulty in swallowing and symptoms referable to the brachial plexus. Upper respiratory infection may be accompanied by infection in the area of the hygroma, with resultant localized abscess formation or blood stream infection.

The differential diagnosis includes: branchial cysts; lipoma; thyroglossal duct cyst; and lymph node enlargement resulting from lymphoma, leukemia or inflammatory changes.

The histopathology reveals numerous dilated cystic spaces lined by endothelium, with or without evidence of smooth muscle. Areas may resemble lymphangioma with cavernous features. Aggregations of lymphoid tissue are found in the interstices of endothelial lined spaces (fig. 10 *A* and *B*). The stroma of the tumor usually contains adipose tissue. In some of the hemangiomatous areas, lymphatics are found which empty into veins.

Treatment. Incision and drainage of the cystic areas is not curative and invites infection. X-ray and radium therapy have been used in selected patients with good effect. Because of the frequent extensive ramifications of the disease, such therapy has limited application. Surgical excision is the treatment of choice, providing the disease is confined to areas of possible dissection. If recurrence develops, it will usually appear within the first postoperative year.

Sclerosing solutions (preferably 25 per cent glucose in young children) will temporarily shrink the cysts, which may make the disease process easier to remove surgically. Strict asepsis must be observed during the injection period to prevent infection. Pressure dressings are placed over the cysts which have been injected, to aid in the obliteration of the cystic cavities. Surgical excision can be carried out with reasonable safety from three to six weeks after the treatment by sclerosing solutions.

In excising the hygromatous mass, the tumor and all of its ramifications must be dissected out. Multiple stage operations are much less frequent since irradiation and sclerosing agents have been used as preliminary procedures in an effort to shrink the process to more reasonable proportions for surgical excision.

Where recrudescence of the disease is noted, x-ray therapy, in moderate dosage, has proved of great benefit. We have observed three such cases

in children varying from two to four years of age and, in all instances, the recrudescence of the disease has been favorably affected.

THYROGLOSSAL DUCT CYSTS AND FISTULAS

There are two types of swelling in the neck related to the thyroglossal tract, namely the pyramidal lobe of the thyroid gland most often located just to the left of the thyroid cartilage and thyroglossal tract cyst. The thyroglossal tract represents the embryologic remnant of the fetal thyroglossal duct, extending from the isthmus of the thyroid to the foramen cecum on the back of the tongue, at the junction point made by the circumvallate papillas. A thyroglossal cyst may be observed at any level between these two points, in the floor of the mouth, at the level of the notch in the thyroid cartilage, or at the level of the thyroid isthmus. The most constant location for such a cyst, however, is to the left of the midline and opposite the notch in the thyroid cartilage. The duct may remain open from the foramen cecum to the skin of the neck, forming a thyroglossal fistulous tract. The presence of the duct or its anomalies frequently is made known first by an infection in the area. On occasion, a thyroglossal cyst may be found to the right of the midline. The cysts vary in size from one to three or more centimeters in diameter. The contents of the cysts are usually mucoid in type. If infection is present, the cyst rapidly increases in size and may be filled with purulent material.

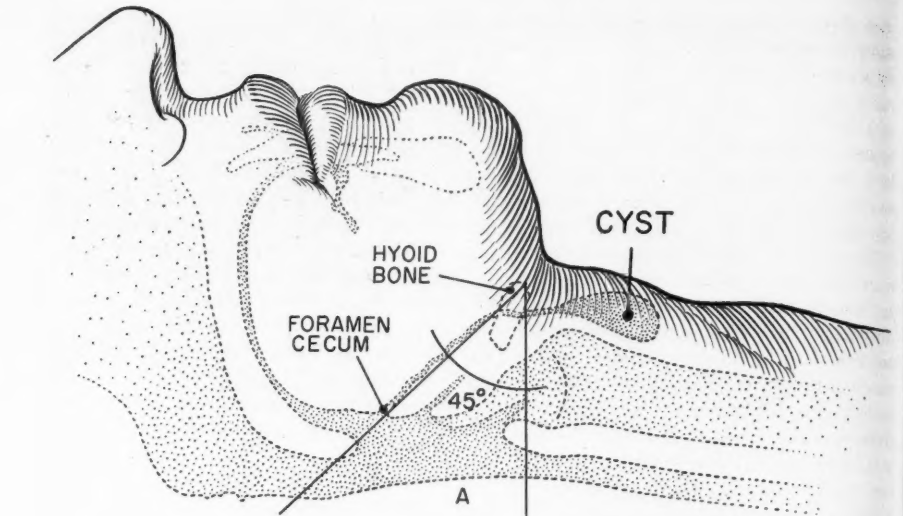
The differential diagnosis includes: follicle or inclusion cysts; lipoma; enlarged lymph nodes near the cricothyroid membrane; and enlarged submental lymph nodes. An important physical finding in differentiating such lesions is the fact that the thyroglossal duct cyst moves vertically with deglutition.

Thyroglossal fistulas are usually near the midline in contradistinction to the branchial fistulas or sinus tracts which are found in the lateral neck.

Histopathologically, the anomalies of the thyroglossal duct tract are lined with stratified squamous epithelium with some tendency at times to show transitional or columnar-type cell morphology. Infection causes desquamation of epithelium and fibrosis. Near the isthmus, thyroid tissue may be found in the tract or in the cyst formation. In one patient, we have seen carcinoma developing in thyroid tissue lining the wall of a thyroglossal cyst well above the isthmus of the thyroid.

Treatment. Complete ablation of the cyst, sinus or fistulous tract is necessary to effect a cure. The technique proposed by Sistrunk has proved to be the operation of choice. A transverse incision of appropriate length (5 to 8 cm.) is made over the cyst, or a bilateral elliptical transverse incision is made about the sinus opening of a fistulous tract. The skin flaps are reflected. The cyst or fistulous tract is followed to its upper extent. If the lesion begins below the hyoid bone, the cyst or fistulous tract is followed to the level of the hyoid bone, where it may be found to pass anterior to it, or to pass through or behind the bone (fig. 11 *A* and *B*). In the latter two instances, it is necessary to remove a segment of the hyoid bone in order to insure complete removal of the tract, and also to facilitate the further dissection of the lesion above the bone. Usually about 1 cm. of the bone is removed with scissors in the case of extremely young individuals, or with bone cutters in adult patients. Traction on the fistulous tract or cyst, or with an Allis clamp on the resected portion of hyoid bone itself, will pull the freed portion of the hyoid bone forward, clarifying the course of the tract above the hyoid bone. It is usually quite small and delicate and requires great care not to pull the tract wall apart. The pressure of the assistant's finger on the base of the tongue through the oral cavity makes for easier coring of the tissue about the sinus tract in the tongue muscle. Should the defect extend into the oral cavity, at the foramen cecum, a purse-string suture of fine catgut will readily close the defect. The musculature of the tongue is then reapproximated with interrupted sutures of either fine catgut or fine black silk. The residual hyoid bone edges are brought together with either fine silk or catgut sutures placed through the bone in children or through the periosteum or surrounding fascia in adults. We have not observed any derangement in the action of the muscles attached to the hyoid bone if the ends of the bone cannot be approximated. A small Penrose drain is placed in the depths of the wound over the hyoid bone. The skin is closed in a routine manner (fig. 11 *C*).

When the cystic tumor presents in the region of the foramen cecum, the tumor is removed through the oral cavity. The tongue is brought



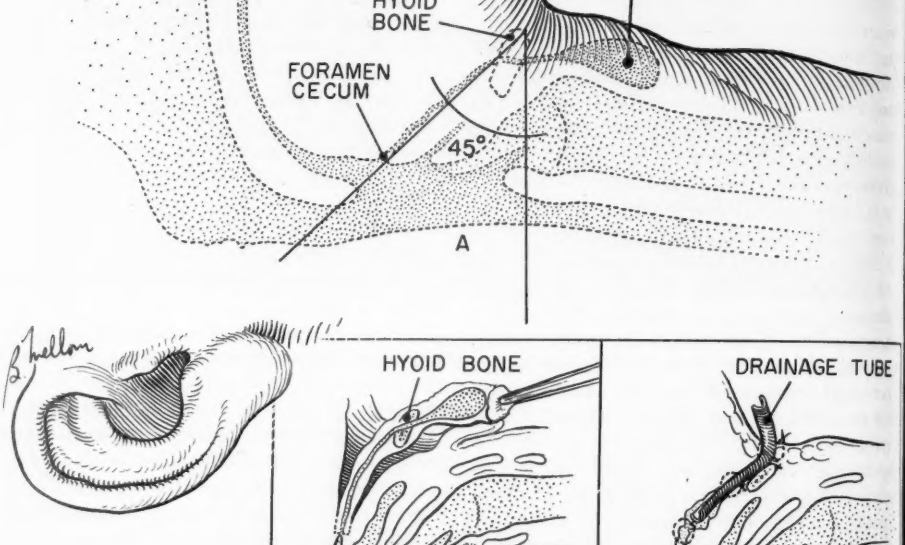


FIG. 11. Diagram (*A*) indicates the obliquity of the angle at which the remnant of thyroglossal duct may traverse the tongue with reference to the hyoid bone. (*B*) Extent of excision of thyroglossal cyst and duct to the area of the foramen cecum at the base of the tongue. Note that a portion of the hyoid bone may be resected if necessary. (*C*) The hyoid bone has been reapproximated and drain inserted. (These drawings reflect points in the operative procedure as originally described by Sistrunk.[39])

forward by means of a silk suture passed through its anterior portion. An elliptical incision is made around the tumor in the long axis of the tongue. Bleeding points are clamped and tied with ligatures of fine catgut, the tumor having been completely excised. The incision in the tongue is closed with interrupted sutures of fine chromic catgut. More recently,[1] Ward has described a submaxillary approach for the surgical removal of large cysts within the tongue substance.

PAPILLARY CYSTADENOMA LYMPHOMATOSUM OF THE PAROTID SALIVARY GLAND

This lesion is also known as Warthin's Tumor or Orbital Inclusion Cyst. It is a multilocular cystic tumor in the region of the parotid gland or about the angle of the jaw, which at times may clinically resemble a branchial cleft cyst and appear somewhat like it microscopically.

Papillary cyst adenoma lymphomatosum constitutes about 10 per cent of all benign parotid neoplasms, occurring in males 10 times more

often than in females. The age distribution is rather widespread, extending from the second to the seventh decades. It is usually a slow growing tumor with an average duration of three years prior to treatment. Patients usually seek advice because of the defect, and experience no symptoms from the tumor. Ten per cent of the tumors may be bilateral and 5 per cent of patients may have multiple lesions in one parotid gland.

The majority of the growths are situated beneath the fascia overlying the parotid gland but the location of the tumor may be quite variable. They have been found superficial to the capsule but attached to it; embedded within the salivary gland tissue; located behind the gland; or lying in the upper neck attached to the tail of the gland. Tumors in this latter location are often confused with branchial cysts or, occasionally, with thyroid adenoma.

Many sources have been suggested to account for the origin of papillary cystadenoma lymphomatosum, including branchial arches, carotid lymph nodes, Eustachian tube, a portion of the cervical sinus, thymic anlage, oncocytic cells, remnants of the orbitoparotid duct, embryonal buccal ectoderm, and heterotopic salivary gland rests situated in lymph nodes adjacent to the parotid gland. No conclusive evidence supports entirely any of these suggested sources of origin.

The differential diagnosis includes: cystic mixed tumors of salivary gland origin; branchiogenic cyst; metastases from silent primary cancer in oral cavity or pharnyx region; carotid body tumor; thyroid adenoma; inflammatory lymph nodes; or lymphoma in cervical lymph nodes.

Preoperative diagnostic studies include a sialogram of the parotid gland and aspiration biopsy. Sialograms have offered us very little help in the diagnosis of this tumor. Aspiration biopsy has been helpful and we do not believe that it enhances the risk of recurrence. It frequently makes it unnecessary to do surgical biopsy before excision.

Warthin's tumor is largely an encapsulated tumor although, occasionally, it may be found infiltrating the parotid substance for a small distance. The capsule is thin, the tumor feels soft and fluctuant (fig. 12). The cut surface is pseudolobulated and contains cysts, from several millimeters to 2 cm. in diameter. The cyst lining is composed of squamous or columnar cell epithelium, with numerous in-foldings overlying aggregtes of lymphoid tissue (fig. 13).

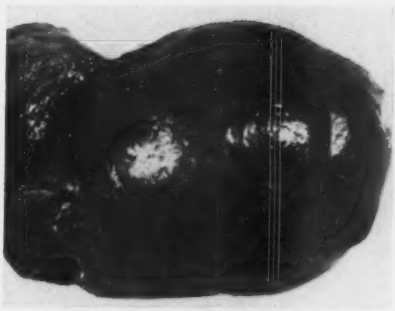


FIG. 12. Warthin's tumor, gross specimen, removed from tail of left parotid gland which extended down into anterior upper cervical zone. Note the glistening capsule and lobulated appearance; small dilated vessels course over the surface of the capsule.

Treatment. The treatment of choice is extracapsular surgical excision of the tumor by blunt and sharp dissection, following surgical biopsy for diagnosis. Excision of noninfiltrating, encapsulated tumors which lie at the edge of the gland or within a short distance of the outer surface may be carried out by making a straight or radial incision over the surface of the tumor. Once the dissection is carried through the subcutaneous tissues and the capsule of the tumor encountered, the growth can be excised by blunt and sharp dissection with a minimum of trauma, both to the parotid gland and to the branches of the facial nerve. When the tumor arises in the posterior or retromandibular portion of the parotid gland, the safest approach is made through a "Y" shaped incision, one branch running upward on the aspect of the lobule and the tragus, and the other running up behind the ear close to the reflection of the skin over the mastoid. The stem of the "Y" is then projected down along the upper anterior border of the sternocleidomastoid muscle. The "Y" incision permits upward retraction of the lobe of the ear and access to the posterior and retromandibular portions, as well as the anterior portion of the parotid gland. This approach may be used to advantage in practically all cases, except for small, encapsulated tumors at the anterior edge of the gland. By exposing the main trunk of the seventh nerve, in all patients with lesions situated in the area, the inadvertent injury of this vital structure is rarely encountered.

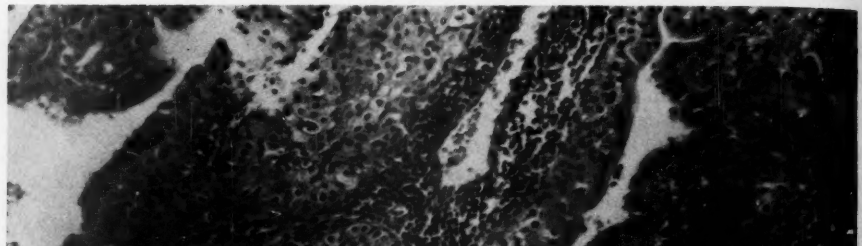


FIG. 13. Microphotograph of papillary cystadenoma lymphomatosum. Note the pseudolobulations about the cystic cavity lined by cuboid to columnar type epithelium which overlays aggregates of lymphoid tissue.

Exposure of the upper border of the posterior belly of the digastric muscle and its attachment to the mastoid process serves as an important landmark. The potential space above the posterior belly of the digastric muscle, near its attachment to the mastoid process, on careful dissection reveals the main trunk of the seventh nerve, after it has emerged from the stylo-mastoid foramen. The trunk of the nerve is usually 3 to 4 mm. in diameter and is readily identified, being at a depth of about 1.5 cm. below the external surface of the mastoid process. Once the trunk of the nerve is identified, any portion of the parotid gland, or any tumor within it, may be readily biopsied and dissected out under direct vision. The wound is drained routinely and the skin flaps approximated by means of interrupted sutures of fine black silk.

The prognosis in treating this disease is excellent. It is considered a benign neoplasm, and no suggestion of malignant transformation has been observed in our experience. Recrudescence due to incomplete surgical excision has been the main complication in treating the disease.

CYSTIC DEGENERATION OF LYMPH NODES INVOLVED BY METASTATIC CARCINOMA, LYMPHOMA, OR INFLAMMATION OF THE LYMPH NODES

The final differentiation of these lesions from some of the other primary cystic tumors we have previously described, is sometimes difficult. In the final analysis it requires aspiration biopsy or formal surgical biopsy. One should exhaust every means of diagnosis before biopsy is done, especially in those patients in whom the definitive treatment might include radical neck dissection at a later date. To do formal excisional biopsy of a neck node as a preliminary study and then ultimately find a carcinoma of the tongue, floor of the mouth, etc., vitiates the survival rate because the lymphatics have been disrupted in the neck loaded with cancer cells. Appropriate cultures and studies of degenerated, inflammatory lymph nodes can be made from aspiration biopsy

material, but if still in doubt, formal surgical biopsy should be carried out.

Metastatic carcinoma involving lymph nodes in the neck may be so extensive as to be inoperable, or the metastases may arise from lesions which are not resectable in continuity with the neck nodes. One is referred to a previous communication by one of us (M.M.C.)[7] on the evaluation of radical neck dissection for cancer about the head and neck.

SUMMARY

Cystic tumors of the neck have been discussed and include:

1. Epidermoid or sebaceous cysts.
2. Branchial cysts.
3. Lymphatic cysts or cystic hygroma.
4. Thyroglossal duct cysts.
5. Papillary cystadenoma lymphomatosum of the parotid gland.
6. Cysts with malignant changes. (Found in epidermoid and sebaceous cysts, rarely in branchiogenic cysts and occasionally in thyroglossal duct cysts from aberrant thyroid tissue in the cyst wall).
7. Cystic degeneration of lymph nodes involved by metastatic carcinoma, lymphoma or inflammation.

The diagnostic features of the benign lesions, and the differential diagnosis, histopathology, treatment and prognosis of each have been outlined.

Malignant changes found in epidermoid or sebaceous cysts, branchial cleft cysts and, occasionally, in thyroglossal duct cysts from thyroid tissue in the cyst wall are discussed. The case for branchiogenic carcinoma is emphasized in some detail.

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PRESACRAL NEURECTOMY AND ITS EFFECTS ON SUBSEQUENT PREGNANCIES*

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The operation of presacral neurectomy is being performed more often in the reproductive period today than ever before and thus this subject is one of ever increasing importance. It is not our purpose to discuss here the technique or the indications for the operation, but rather to focus attention on the altered pattern of labor in patients previously subjected to presacral neurectomy.

In a fairly comprehensive review of the literature we found 262 case observations of postneurectomy labor reported by various authors.[2-6, 8, 10, 15, 16, 18-21, 26, 27, 30, 35, 37, 38, 40-44, 46] A great many were reported incompletely and many of the authors depended upon second hand information. It is not surprising, therefore, that there has been great disagreement on the effects of presacral neurectomy on subsequent pregnancies. Cotte[11, 12] himself reported that in over 50 cases of postneurectomy pregnancies and labor the normal pattern was followed. Dumont[20] however, closely observed many patients operated upon by Cotte and came to quite different conclusions. As recently as 1949 Browne[7] stated that "labor following any form of nerve resection was neither abnormal, nor easier, nor more difficult than the average."

Presacral neurectomy for the relief of pelvic pain was performed by Jaboulat[31] 59 years ago. This was the first attempt at the interruption of the sympathetic afferent pathways from the pelvic viscera and although not entirely successful, led the way to freedom and understanding for many unfortunate women hitherto branded as hypochrondriacs or neurotics.

In 1899 Ruggi[39] reported a number of cases in which pelvic pain was relieved by utero-ovarian plexus resection; however, it was the work of Leriche[33] in 1921 which really established the procedure, although his main aim was to relieve the pain of pelvic malignancy.

In 1925 Cotte found that resection of the presacral nerve of Latarjet gave relief in cases of primary dysmenorrhea. He was careful to lay emphasis on the exclusion of ovarian dysmenorrhea before performing this operation, and reported a failure rate of only 1 per cent in his first 200 selected cases. By 1949, in a series of 1500 cases, a failure rate of 2 per cent was reported by Cotte.[13] In the past, American gynecologists were hesitant in accepting this operation and there was a general feeling that some European workers were rather liberal in their choice of indications.

Herrmann is generally accredited with being the first to perform presacral neurectomy in this country, and, in association with Fontaine,[24] afforded apparent relief in 200 cases of pelvic plexaglia and primary dysmenorrhea.

In recent years a very large number of these operations have been performed and reports are now manifold, including those of Adson and Masson[1], DeCourcy[17], Wetherell,[45] Flothow,[23] and others. The operation is now a safe and well established therapeutic procedure which is enjoying ever increasing popularity. It has been found especially effective in the treatment of primary dysmenorrhea and also in combination with other procedures in secondary dysmenorrhea. As pointed out by Ingersoll and Meigs,[30] selection of cases and careful technique are the most important factors in achieving satisfactory gynecologic end results.

A great deal has been written about the anatomy of the pelvic autonomic nervous system and many excellent articles are available, particularly the classic work of Curtis and his associates.[14] It is our intention here only to present points immediately pertinent to the subject under discussion.

The term "presacral nerve" is misleading because it is not a nerve, but a plexus, in about 80 per cent of the cases. Moreover, only a small portion of it is presacral, for it lies in most patients over the anterior surface of the fifth lumbar and upper part of the first sacral vertebra. The presacral nerve has been more correctly named the

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"superior hypogastric plexus" by Hovelacque.[29] We have decided to retain the name of presacral nerve because it is more commonly known.

Elaut's[22] dissections reveal four main variations of this plexus: (1) 58 per cent of the patients showed atypical plexuses lying between the common iliac arteries, (2) 24 per cent had a single distinct nerve trunk, (3) 16 per cent had parallel nerve trunks, and (4) 2 per cent showed an arch-shaped plexus. Especially with the arch-shaped plexus, the surgeon may retract one arm of the arch with the sigmoid and resect only one portion of it in the mistaken belief that he is dealing with a single nerve trunk. We believe that these points are important and that postoperative failures, in carefully selected cases, as well as in many patients in whom painful labor has been reported after this operation, are the result mainly of the failure of the surgeon to appreciate these anatomic possibilities.

THE PATHWAYS OF LABOR PAIN

The main credit for the establishment of the pathways of labor pain goes to Clelend[9] who in 1933 first determined these pathways in the fundus and birth canal in animals. After a study of comparative anatomy, he concluded that afferent impulses from the human fundus should enter the cord at T-11 and T-12. He also deduced that pain from cervical dilatation and from vaginal and perineal distention should enter the cord at sacral segments two, three, and four. Using nupercaine and procaine he carried out paravertebral blocks at T-11 and T-12 and successfully relieved fundal and corporeal pain. By means of caudal anesthesia he then blocked the sacral nerves and this resulted in complete relief from labor pain.

The earliest sign of true labor in the average pregnant woman is the establishment of rhythmic contractions of the uterus—these contractions being associated with pain to a greater or lesser degree. In early labor this pain originates from pressure on the nerve endings between the muscle fibers of the uterus; as labor progresses the contractions increase in severity, duration and frequency. Pain increases correspondingly with progressive retraction of the individual muscle fibers as the passenger is propelled through the birth canal. This pain has been found to be transmitted by way of the sensory fibers from the corpus and fundus of the uterus to the large circumcervical plexus of Frankenhauser, and thence *via* the hypogastric plexuses through the paravertebral sympathetic chain at the level of the second and third lumbar vertebras. The pain impulse continues without synapse cephalad. Traversing the gray rami communicantes of the 11th and 12th thoracic and possibly the first lumbar nerve, it enters the communicating system of these dorsal root ganglia with the preganglionic afferent system in the lateral spinothalamic tract to reach the thalamic pain center and its cortical radiations.[37] This is undoubtedly the most important pathway of fundal and corporeal labor pain.

The second component of labor pain is associated with dilatation of the cervix and is most commonly characterized by backache. Here the pain impulses are transmitted through the nervi erigentes of the parasympathetic sacral outflow to sacral cord segments, two, three, and four.

The third component of labor pain is that arising from distention of the vagina and perineum. These pain impulses are transmitted by way of the pudenal nerve or its perineal and hemorrhoidal branches to the second, third, and fourth sacral cord segments. Klink[32] has shown that in 50 per cent of patients the inferior hemorrhoidal nerve arises from the sacral plexus, independent of the pudenal nerve.

Two other components of labor pain may be present. Anselmino and Plaskuda[2] believe that a fourth pathway is present by which pain impulses pass from the cervix *via* Frankenhauser's plexus and directly to the sacral sympathetic chain to enter the cord at thoracic segments 11 and 12.

A fifth and relatively unimportant pathway is that from the cornu by way of the ovarian sympathetic plexus to the spinal cord.

CASE REPORTS

In our series of cases (table 1) the following points should be made:

1. All patients had presacral neurectomy performed by the senior author (W. J. R.) after conservative therapy had proved ineffectual. In addition, a purely psychoneurotic basis had been excluded using the "stilbestrol test" as described by Ingersoll and Meigs.

2. Routine sedation at our institution consists of: Seconal 3 gr., Demerol 100 mg., and scopolamine 1/150 gr., given parenterally.

3. All surgical inductions in this series consisted

TABLE 1

Summary case reports

Patient	Parity	First Stage	Second Stage	Dilation; Sedation	Delivery	Postpartum	Labor
1. T.B., age 24; infant, 6 lb. 13 oz.	0-0-0-0	Painless	Sacral rectal pressure	6 cm.; routine sedation	Low forceps episiotomy, pudendal 12/31/49	Normal uneventful	6 hr.
2. D.K., age 27; infant, 7 lb. 3 oz.	0-0-0-0	Terminal rectal pressure	Rectal pressure	Fully; routine sedation	Low forceps episiotomy, saddle, 3/14/50	Normal uneventful	2 hr.
3. A.B., age 39; infant, 7 lb.	2-0-0-2	Painless artificial rupture of membranes	Vaginal pressure	6–7 cm.; routine sedation	Low forceps episiotomy, saddle, 6/23/52	Slight afterpains	3 hr., 45 min.
4. T.B , age 27; infant, 6 lb. 9 oz.	1-0-0-1	Low back ache, artificial rupture of membranes	Rectal pressure	6 cm.; routine sedation	Low forceps episiotomy, saddle, 7/25/53	Normal uneventful	1 hr., 55 min.
5. M.K., age 25; infant, 6 lb. 5 oz.	0-0-1-0	Low back ache	Sacral rectal pressure	8 cm.; routine sedation	Low forceps episiotomy, saddle, 3/5/55	Normal uneventful	4 hr., 15 min
6. A.S., age 21; infant, 2 lb. 14 oz. (lived)	0-0-0-0	Painless	Slight rectal pressure	None; (30 weeks pregnant)	Low forceps episiotomy, saddle, 11/14/56	Normal uneventful	4 hr., 30 min.
7. M.S., age 26; infant, 7 lb. 6 oz.	0-0-0-0	Rectal pressure	Perineal pain	5 cm.; routine sedation	Low forceps episiotomy, saddle, 12/6/56	Normal uneventful	10 hr., 5 min.
8. B.I., age 28; infant, 7 lb. 15 oz.	0-0-0-0	Painless artificial rupture of membranes	Rectal pressure	6 cm.; routine sedation	Low forceps episiotomy, saddle, 12/28/56	Normal uneventful	3 hr., 40 min.
9. A.B., age 43; infant 7 lb.	3-0-0-3	Low back ache, artificial rupture of membranes	Slight rectal pressure	7 cm.; routine sedation	Low forceps episiotomy, saddle, 1/10/57	Normal uneventful	2 hr., 30 min.
10. J. B. age 33; infant 6 lb. 11 oz.	2-0-0-2	Painless	Slight backache	8 cm.; routine sedation	Low forceps episiotomy, saddle, 1/14/57	Normal uneventful	4 hr.

of artificial rupture of the membranes, preceded by a high hot enema.

4. Saddle spinal block or pudendal block is used unless contraindicated.

Primigravida are "saddled" at full dilatation of the cervix with the vertex on the perineum while multigravida are "saddled" at approximately 9 cm-cervical dilatation with the vertex at plus one station.

Case 1. Mrs. T. B., age 24, para 0-0-0-0. This patient had presacral neurectomy performed in October 1947. She became pregnant and her E.D.C. (estimated day of confinement) was January 6, 1950. After an uneventful prenatal course she was admitted to the hospital on December 31, 1949 with bloody show but no pain. At 12 noon painless contractions were noted to be occurring regularly about every 7 min., each lasting 30 sec., and at this time the head was engaged and the cervix dilated 1 cm. The patient was sedated at 4:50 p.m. when the cervix was dilated 6 cm., and she was delivered under pudendal block, by prophylactic low forceps and central episiotomy, of a male child weighing 6 lb. 13 oz. at 5:44 p.m. The placenta and membranes were expressed intact at 6:00 p.m. and the blood loss estimated to be about 150 cc. The puerperium was uneventful.

Case 2. Mrs. D. K., age 27, para 0-0-0-0. This patient, a para 0-0-0-0, was married in 1948 and was first seen in 1949 complaining of primary dysmenorrhea. Conservative therapy had failed; therefore, after a "stilbesterol test," presacral neurectomy was carried out. The patient subsequently became pregnant with an E.D.C. of March 10, 1950. Her prenatal course was uneventful. On March 14 at 2:45 a.m., stating she had some show, she called the obstetrician. She was told to await the onset of pains. Shortly thereafter she phoned again and was told at this time to go to the hospital, despite the fact that she had no pains but simply bloody show and some pressure in the pelvis. This patient had no pains whatever on admission. Examination on admission at 3:45 a.m. revealed a vertex presentation at station plus two with cervix fully dilated. Fortunately, the obstetrician had arrived at the hospital just prior to the patient's admission, although up to this point the presacral neurectomy had been forgotten. The patient was given routine sedation. At 4:00 a.m. she was delivered, by means of low forceps and central

episiotomy, of a 7 lb. 3 oz. active female child under saddle block anesthesia. The placenta and membranes were expressed intact at 4:15 a.m. with an estimated blood loss of approximately 150 cc. The postpartum course was uneventful and the patient has had no recurrence of her dysmenorrhea.

Case 3. Mrs. A. B., age 39, para 2-0-0-2. This patient had two full term normal deliveries, involving the usual amount of pain, prior to her presacral neurectomy. The operation was performed on January 22, 1948 after the usual preliminary investigation and therapy. Her dysmenorrhea was very much ameliorated following this procedure. The patient was seen again in her third pregnancy with an E.D.C. of June 23, 1952. Her prenatal course was uneventful. On June 23, vaginal examination revealed vertex presentation at station 0 with cervix 60 per cent effaced and dilated 2 cm. The patient was advised to accept surgical induction, and after admission to hospital artificial rupture of the membranes was carried out at 4:15 p.m. At 5:00 p.m., contractions lasting 20 sec., every 10 min. were noted and thereafter the contractions increased but the patient had no noticeable abdominal pain whatever and only slight rectal pressure was felt toward the end of the first stage. Sedation at 7:00 p.m. was given when the cervix was 80 per cent effaced and dilated 6 cm. with the vertex at station plus one. At 8:00 p.m. the patient was delivered, by low forceps and central episiotomy under saddle-block anesthesia, of a 7 lb. child. At 8:10 p.m. the placenta and membranes were expressed intact with the blood loss estimated at 150 cc. The postpartum course was uneventful and the patient has had no recurrence of her dysmenorrhea.

Case 4. Mrs. T. B., age 26, para 1-0-0-1. This patient's first pregnancy is reported as case no. 1. She again became pregnant with E.D.C. of July 23, 1953. Her prenatal course was again uneventful and she was admitted for surgical induction at her own request on July 25, 1953 after office examination revealed favorable circumstances. Admission examination at 2:00 p.m. confirmed the presence of an engaged vertex with cervix soft, 40 per cent effaced and dilated 1 cm., and artificial rupture of the membranes was carried out. At 3:10 p.m. the patient complained of low backache but had no abdominal pain, and vaginal examination at this time revealed that the cervix was dilated 6 cm. with vertex at station plus one and sedation was given. Labor progressed rapidly with her only complaints being of low sacral ache and rectal pressure. Low saddle block anesthesia was given at 3:40 p.m. The patient delivered at 3:50 p.m., by low forceps and central episiotomy, of a 6 lb. 9 oz. active male child. The placenta and membranes were expressed spontaneously at 4:00 p.m. and the estimated blood loss was approximately 100 cc. The postpartum course was entirely uneventful. This patient has had no recurrence of her dysmenorrhea after either of these pregnancies.

Case 5. Mrs. M. K., age 25, para 0-0-1-0. This patient had a presacral neurectomy performed in 1951 for severe primary dysmenorrhea which had not responded to conservative therapy. After her operation she was markedly improved and became pregnant in November 1951. On January 30, 1952 the patient was admitted to the hospital with vaginal bleeding which increased and with the association of slight terminal sacral ache and an urge to defecate—she aborted on February 2, 1952. Following this pregnancy there was no recurrence of her dysmenorrhea.

The patient was again seen on October 1, 1954 at which time she was found to be pregnant with an E.D.C. of March 5, 1955. Her prenatal course was uneventful and she was admitted to hospital on March 5, 1955 at 11:30 a.m. because of "bloody show." At that time no regular contractions were noted and examination revealed vertex engaged with the cervix approximately 50 per cent effaced and dilated about 1 cm. At 6:00 p.m. on March 5 it was noted that the patient was having painless contractions lasting about 20 sec. every 10 min. At 7:00 p.m., with the cervix dilated 4 cm. and 70 per cent effaced, the patient complained of low backache. At 9:00 p.m. she was sedated, at which time the cervix was dilated 8 cm. and 80 per cent effaced. At 10:00 p.m. an active 6 lb. 5 oz. female infant was delivered by means of low forceps and central episiotomy under saddle-block anesthesia. At 10:15 p.m. the placenta and membranes were expressed intact. The estimated blood loss was 150 cc. This patient had no abdominal pain but sacral ache appeared early in the first stage and also rectal pressure was felt in the second stage. The patient had not voided 8 hours after delivery so she was catheterized. Thereafter the patient voided spontaneously and we believe that at the time of catheterization this was not really necessary. Her postpartum course was uneventful. Following delivery, the patient has had no recurrence of dysmenorrhea and is again pregnant and is due in March 1957.

Case 6. Mrs. A. S., age 21, para 0-0-0-0. This patient was first seen at the age of 19 years with severe primary dysmenorrhea. Conservative therapy at that time was instituted but was ineffectual and a presacral neurectomy was performed in January 1955. Following this operation the patient had no further dysmenorrhea. She was married and conceived within the next 18 months.

Her E.D.C. was February 2, 1957. Her prenatal course was uneventful until November 9, 1956 when she was admitted to the hospital with painless bleeding. Placentography showed no placenta prævia but a footling breech presentation was noted. Gentle sterile pelvic examination was performed and the cervix was found to be 50 per cent effaced and dilated approximately 1 cm. Slight bleeding persisted but the patient had no contractions until November 14, at 1:00 p.m., when contractions lasting 20 sec. every 10 min. were noted. Careful vaginal examination revealed a vertex presentation with the cervix dilated 2 cm. and 50 per cent effaced. It was believed at this time that premature labor was inevitable but "Releasin" therapy was started. Despite this, the patient's contractions increased and at 3:00 p.m. she was having strong contractions lasting 40 sec. every 5 min. These were completely painless and in view of the prematurity of the child, no sedation was given. At 5:10 p.m. the patient's cervix was fully dilated with the vertex at station plus two in an occiput anterior position. At 5:20 p.m., under saddle-block anesthesia, a 2 lb. 14 oz. active female child was delivered by low forceps and central episiotomy. The placenta and membranes were expressed intact at 5:30 p.m. with an estimated blood loss of 200 cc. It should be noted that this patient had no painful abdominal cramps and no sacral ache or rectal pressure. She voided spontaneously a few hours after delivery and her postpartum course was completely uneventful.

Incidentally, the baby was discharged from the hospital two months later in good condition and weighing 5 lbs.

Case 7. Mrs. M. S., age 26, para 0-0-0-0. This patient was seen in October 1952 with complaint of severe primary dysmenorrhea of 10-years duration. Conservative measures had previously been ineffectual and a presacral neurectomy was performed on January 22, 1953. Postoperatively, the patient had complete relief from her dysmenorrhea.

On April 30, 1956, the patient returned pregnant with an E.D.C. of December 10, 1956. Her prenatal course was uneventful. She was admitted to the hospital on Dec. 7, 1956 because of slight bloody show. At 2:00 p.m. abdominal examination revealed a vertex presentation with the head engaged in left occiput transverse position. Vaginal examination revealed the cervix dilated 1 cm. to 2 cm., 60 per cent effaced, and at this time contractions lasting about 20 sec. every 10 min. were noted. At 7:45 p.m. the cervix was dilated 5 cm. and approximately 80 per cent effaced with the vertex at station plus one. The patient was having contractions lasting 30 sec. every 4 min. but these were completely painless. Sedation was given at this time. At 11:55 p.m. an active 7 lb. 6 oz. infant was delivered by low forceps and central episiotomy under saddle-block anesthesia. The placenta and membranes were expressed intact at 12:05 a.m. This patient at no time had abdominal pain but complained of sharp shooting pains in the rectum and of perineal pressure at the end of the first stage and during the second stage of labor. She voided spontaneously 8 hr. after delivery and her entire postpartum course was uneventful. Since delivery the patient has had one menstrual period and this was quite painless.

Case 8. Mrs. B. I., age 28, para 0-0-0-0. This patient was married 4 years prior to her presacral neurectomy on February 11, 1953. She subsequently became pregnant with an E.D.C. of December 31, 1956. Her prenatal course was uneventful. On December 26, 1956, vaginal examination revealed a vertex presentation at station minus 1, cervix dilated 1 cm., soft and effacing. On December 28 the patient was admitted for elective induction of labor, because at this time the cervix was found to be dilated 1 cm., soft and 50 per cent effaced, with the vertex at station 0. The indication for induction was the extreme fear of this patient that she might deliver unattended at home. At 10:00 p.m. on December 28 artificial rupture of the membranes was performed. At 11:35 p.m. the patient began to have regular contractions lasting about 30 sec. every 5 min., although she was not conscious of pain. At 1:00 a.m. on December 29 the cervix was dilated 6 cm., 90 per cent effaced, with the vertex at station plus one. Although the patient had no conscious pain, but because of her marked anxiety, sedation was given at this time. At full dilation of the cervix, the patient was conscious of rectal pressure only. At 3:10 a.m. she was delivered, by means of low forceps and central episiotomy under saddle-block anesthesia, of an active child weighing 7 lb. 15 oz. The placenta and membranes were delivered spontaneously and intact at 3:15 a.m. with an estimated blood loss of approximately 150 cc. The postpartum course was uneventful.

Case 9. Mrs. A. B., age 43, para 3-0-0-3. This patient returned pregnant with an E.D.C. of January 18, 1957. Her pregnancy was again uneventful. On January 3, 1957 the patient requested induction of labor, being afraid that she might deliver at home and unattended. At this time the vertex was not engaged and the cervix was thick and fairly firm, so her request was refused. Examination on January 10 revealed cervix dilated 1 cm. to 2 cm. and soft, and approximately 60 per cent effaced. The patient was then admitted to the hospital and surgical induction carried out. At 3:00 p.m. she was found to be having regular con-

tractions lasting approximately 20 sec. every 5 min. At 4:30 p.m. the cervix was dilated 6 cm. and approximately 80 per cent effaced. Sedation was given, although at that time she had no pain whatsoever. When the cervix was dilated 9 cm., she complained of rectal pressure and some sacral ache but had no abdominal cramps. At 5:15 p.m. the patient was delivered, by means of low forceps and central episiotomy under saddle-block anesthesia, of a 6 lb. 15 oz. active infant. At 5:30 p.m. the placenta and membranes were expressed intact with an estimated blood loss of 200 cc. The postpartum course was uneventful apart from some "after pains" on the second day. This patient has had no further menstrual periods since her delivery. Her six-week check-up was normal.

Case 10. Mrs. J. B., age 33, para 2-0-0-2. The patient was first seen in January 1949 with complaint of dysmenorrhea of 18-years duration and becoming worse despite marriage and conservative therapy. Her first two children were born with the normal amount of pain and after each pregnancy her dysmenorrhea returned. She was given "two stilbesterol tests" and thereafter was considered suitable for a presacral neurectomy. The operation was performed on June 10, 1949. After operation she had complete freedom from her dysmenorrhea.

On September 14, 1956 the patient returned pregnant with E.D.C. January 22, 1957. Her prenatal course was uneventful except for some pelvic pressure in the last week of pregnancy. On January 4, 1957, she was admitted to the hospital because of "bloody show" although she had no pains. At 6:30 a.m. examination revealed regular contractions lasting 20 sec., every 10 min. Vaginal examination at this time disclosed a vertex at station minus one with cervix dilated 1 cm. to 2 cm. and 60 per cent effaced. At 9:30 a.m. the cervix was dilated 8 cm. and 80 per cent effaced, with the vertex at station plus one. The patient complained of sacral ache and slight rectal pressure. These increased as labor entered the second stage. At no time did the patient have any abdominal pain. At 10:20 a.m. she was delivered of an active 6 lb. 11 oz. male child by low forceps and central episiotomy under saddle-block anesthesia. At 10:30 a.m. placenta and membranes were expressed intact with estimated blood loss of 150 cc. On February 26, 1957, the patient returned for a six-week check-up and stated, "If you could guarantee any future pregnancies as painless as this one, I would be back every year."

DISCUSSION

In a fairly comprehensive review of the literature we found observations made on a total of 262 cases and these, together with our own 10 personally observed cases, prove the basis for our discussion. In reviewing the part played by the presacral neurectomy alone, in comparison with presacral neurectomy and other procedures, Dumont[19] in i951 showed that presacral neurectomy was the only significant factor in attenuation of first stage labor pain in subsequent deliveries.

The most important paper to date on the attenuation of first stage labor pain is that of Dumont[20] (1952) who reported on 124 postneurectomy pregnancies. In reviewing our own 10 carefully observed pregnancies, in patients previously subjected to presacral neurectomy, we are led to the following conclusions which we will correlate with the findings of other authors:

1. Pregnancy was normal in all patients. It is generally accepted that presacral neurectomy has no specific effects on the course of pregnancy.

2. The onset of labor is insidious, the patient often does not believe that she is in labor (this was especially evident in Mrs. D. K., a primigravida who arrived at the hospital fully dilated). This has also been the experience of other authors. Ingersoll and Meigs[30] mentioned two primigravidas in their series who delivered without obstetric assistance since they did not know they were in labor until the baby's head was on the perineum.

3. First stage labor pain appeared definitely attenuated in all patients and indeed was completely absent in two. Wetherell (1935),[44] Donaldson (1936),[18] Bittmann (1938),[4] Darget and Mahon (1943)[15] and Browne (1949)[7] did not find this to be their experience, but most other authors apparently agree that this attenuation of first stage labor pain is the most striking feature of postneurectomy pregnancy.

4. Second stage labor pain is in our opinion also reduced as compared with normal controls. We must qualify this by stating that this observation is based on the primigravida in our series because many of our multigravida were "saddle" at 9-cm. cervical dilation. Another point is that routine sedation was given to our patients at an average of 6-cm. to 7-cm. dilation of the cervix, even though the pain at that time was not severe in our primigravid patients. It was considered that this would allay anxiety and reduce the terminal first stage and second stage pain. When necessary, as in the case of Mrs. A. S., this

sedation can be withheld with relatively little discomfort to the patient. The veracity of this statement is borne out by the examination of Rutherford's detailed report in which apparently five of his own seven patients, who had vaginal delivery, had no sedation whatever until they were fully dilated, and even then no potent analgesics were given.

Anselmino and Plaskuda[2] make no mention of their patients requiring sedation in the first stage of labor.

In a control series of 100 non-neurectomized patients of the physical makeup, age and parity comparable to our patients, it was found the primigravida generally required sedation at 3-cm. and the multigravida at 4-cm. to 5-cm. dilation.

5. Inertia was not seen in any of these patients. Inertia was reported by Blinick (1947)[6] in observation of two deliveries following presacral neurectomy with resection of one ovary and multiple myomectomy. We believe that the latter procedure offers a more logical explanation for the inertia than does the presacral neurectomy. It is our impression that precipitate labor is a much more common feature subsequent to presacral neurectomy than is uterine inertia.

6. Duration of labor in primigravida patients in our series of cases was about 5 hr. as compared with 13 hr. in the control series of 100 cases. Duration of labor in the multigravida was just over 3 hr. as compared with 8½ hr. in the control cases. The more rapid course of labor in these patients probably results from the removal of sympathetic inhibition of labor and the breaking of the "tense mind—tense cervix cycle." In assessing the intensity of pain and duration of labor in the multigravida patient, the progressive diminution of both of these features in the average non-neurectomy patient must be borne in mind.

7. No malpositions were found in our patients. Anselmino and Plaskuda (1950) reported two malpositions in their series of 9 patients, and they explained this high incidence on the basis of relaxation of the pelvic floor musculature causing disturbance of the normal vertex rotation mechanism. They state that this might be expected following presacral neurectomy. Since it is generally agreed that the presacral plexus has nothing to do with the nerve supply of the pelvic floor musculature, we cannot agree with their reasoning. In view of the otherwise excellent nature of their paper we wonder if we have not misunderstood them on this point. Although all our patients were delivered by prophylactic low forceps, this was not the result of malposition or mal descent, but merely because this is the usual mode of delivery in our institution.

8. The third stage in all our patients was normal, with expression of an intact placenta and membranes and with normal blood loss. We have found no reference to postpartum hemorrhage attributable to presacral neurectomy in the literature.

9. The postpartum course was uneventful in all 10 patients. One of the four multigravida complained of "after pains" and, although no conclusion can be drawn from this, we were interested in this aspect. The patient in a case reported by Anselmino and Plaskuda[2] complained of "after pains" although her labor also had been practically painless.

10. In all these cases of successful presacral neurectomy for dysmenorrhea there was no recurrence of the dysmenorrhea following pregnancies.

Let us now consider the groups of patients who are typical and atypical according to the pattern which we have described.

A. The typical labor pattern after presacral neurectomy. Many aspects of the labor pathways of parturition are not completely understood. It is generally accepted that until the cervix nears full dilation the main components of pain are of fundal and corporeal origin, and the main pathway of pain impulses is through Frankenhauser's ganglia and then by way of the presacral nerve to enter the cord at the 11th and 12th thoracic segments.

It has been demonstrated by Cleland (1933),[9] Anselmino, Plaskuda and Stewens,[3] Lull and Hingson[34] and others, that this pathway can be blocked by paravertebral block at the 11th and 12th thoracic segments, by 2nd and 3rd lumbar sympathetic blocks, by low lumbar aortic plexus block, by peridural 11th thoracic through 1st lumbar block, by ascending caudal block or by saddle spinal block anesthesia. Furthermore, within the presacral nerve a significant number of thinly myelinated nerve fibers of the type shown experimentally to be associated with pain transmission have been demonstrated. Therefore, would we not expect marked reduction in first

stage labor pain in the adequately neurectomized patient?

As regards the late first stage pain, Head (1896)[25] basing his observations on the concept of referred pain stated that, during cervical dilation, pain is felt over the sacrum and coccyx and the second, third, and fourth sacral dermatome areas.

In our series, sacral ache was a common complaint at the end of the first stage and this too had been reported by others. Here the second pathway from Frankenhauser's ganglia and by way of the afferent thinly myelinated fibers carried in the pelvic nerves (S2, S3, and S4) is probably involved. Alternatively, some fibers may pass from Frankenhauser's ganglia to the sacral ganglia and thence into the cord at the 11th and 12th thoracic segments. The late first stage pain is probably not the result of cervical dilation but of pressure on cervical nerve endings by the presenting part of the fetus and, where this is not marked, the entire first stage labor may be painless. Evidence for this is found in the presence of pain on forceable dilation of the cervix.

In the second stage the sensations of rectal discomfort and perineal distention were those most commonly observed in our series, and also in most cases in the literature where the second stage was carefully observed. These sensations arise from pressure caused by the descent of the presenting part on the rectum and pelvic musculature, as well as by the distention of the vagina and perineum. Here the main pathways are probably by way of the pelvic nerves and sacral somatic nerves, particularly the internal pudendal.

To date there has been a general tendency to consider the second stage of labor in the neurectomized patient as being normally painful. In our series of cases although the most marked attenuation of pain occurred in the first stage, the total second stage pain appeared also reduced. This of course can only be inferred in the primigravid patients who did not receive saddle-block anesthesia until the head was on the perineum.

In reviewing Anselmino and Plaskuda's series of carefully followed cases, we believe that they would agree with us, although they make no special point of this in their conclusions. In the second stage of labor, pain results from the uterine cramps as well as from the distention of the birth canal and perineum. The uterine component of second stage pain is eliminated by the interruption of the presacral nerve, and we should expect that total second stage pain would be reduced to some degree. To the diminution of pain expected in the typical case we would add the shortened duration of the first stage of labor and consequently of total labor. This we consider is probably the result of the removal of sympathetic inhibition abolishing the "tense mind—tense cervix cycle" and improving the physiologic polarity of the uterus. The autonomy of the uterine contractions has been established by anesthetic blocking and by observation of patients with cord damage (Hingson and Hellman).[28]

B. The atypical labor pattern after presacral neurectomy. We note in reviewing the literature that not all cases described fall into our typical pattern and for this many explanations are available:

1. Incomplete presacral neurectomy will not only fail to relieve dysmenorrhea but will also tend to a normal intensity of labor pain as well as normal duration of labor. Indeed, Dumont[20] in 1952 found attenuation of labor pain in only 25 per cent of 20 patients who had unsuccessful presacral neurectomy in contrast with attenuated pain noted in 37 per cent of 95 normal controls. Elaut's[22] studies in 1932 give ample explanation as to why incomplete presacral neurectomy may occur. We agree with Dumont that this is probably the most important reason for painful contractions in postneurectomy patients.

2. Regeneration of the presacral nerve may occur, or cicatricial neuroma formation as postulated by Dumont probably explains some cases. Provided that at presacral neurectomy the entire plexus is dissected out and a 4-cm. segment is removed, and particularly if Wetherell's technique is followed, this group can be reduced to a minimum. It may be mentioned here that even in some patients who are not relieved of their dysmenorrhea, as in cases where a psychoneurotic basis has not been excluded, attentuated pain in labor may still be expected, provided that adequate presacral neurectomy has been performed.

3. Although adequate presacral neurectomy may have been carried out, the quantitative transmission of pain impulses may be subject to

individual variations and, as Held in 1943 has suggested, abnormal predilection of impulses for certain pathways may occur in some individuals.

It seems that to some degree the following afferent pathways may transmit pain impulses even in the first stage of labor, although we must emphasize that their influence is small in the average patient:

(a) A pathway through Frankenhauser's ganglion to the sacral ganglia and thence to enter the cord at thoracic 11th and the 12th segments. These ganglia are resected unilaterally or bilaterally, together with presacral neurectomy in Held's operation. Anselmino and Plaskuda were impressed that there was an ever greater attenuation of first stage labor pain in these patients than in those who had simple presacral neurectomy, and they consider that this is an important pathway in the first stage of labor.

(b) A pathway through Frankenhauser's plexus and the pelvic nerves (parasympathetic) to sacral cord segments two, three and four.

Magin[35] in 1949 quoted three cases following Richers operation (presacral neurectomy plus resection of the pelvic nerve) where the first stage of labor was painless and somewhat shorter than normal. This route, like the above, probably becomes more important towards the end of the first stage of labor, where pressure on the nerve endings of the dilating cervix, as well as pressure on the adjacent viscera, particularly bladder and rectum, set up impulses which would take this pathway.

(c) A pathway from the cornua and fundus of the uterus passing through the utero-ovarian plexus to the lumbar sympathetic chain. These fibers are of little practical importance and many deny their existence but they may contribute inordinately to first stage labor pain in some patients.

(d) A pathway by way of the periarterial sympathetic fibers which may reach the cord without passing through the presacral plexus. Leriche[33] claimed to have cured dysmenorrhea by periarterial sympathectomy alone. It is worth mentioning here that a sheaf of periarterial sympathetic fibers may bypass the presacral plexus by ascending behind the aorta and so escape detection by even the most meticulous operator.

4. Many authors have not personally observed all their reported cases and have largely based their conclusions on the observations of others.

IMPORTANT PRACTICAL ASPECTS

From all that has gone before one may rush to the conclusion that the benefits of presacral neurectomy in labor are incontestable. There are many dangers inherent in this attitude, for only the careful observation of the patient by the obstetrician and hospital staff, and alertness to the probability of an altered labor pattern, will avert the occasional catastrophy in this increasing group of patients.

1. Abortion. This complication does not appear to occur any more frequently than in normal patients. One of our patients (Mrs. M. K.) aborted almost painlessly at 8 weeks. Anselmino and Plaskuda (1950)[2] reported two cases in which the patients aborted almost painlessly at about 16 and 20 weeks, respectively. Rutherford (1942)[40] reported that one patient in his series "silently aborted thrice at two and four months in ideal fashion." Silent abortion may be ideal as long as the patient is under supervision in the hospital, but we have even seen cases of spontaneous abortions in shock because the patient has chosen to ignore early bleeding and cramps. Few physicians would turn a patient of inevitable abortion away from the hospital because of the hazard of hemorrhage, but is not this hazard also present in the case of the silent inevitable abortion?

2. Antepartum hemorrhage. This complication may occur in these as well as in other pregnant patients. There may be increased difficulty in differentiating placenta praevia from abruptio placentae, but, since the patient will usually call her obstetrician under these circumstances, and most authorities agree that all patients with significant antepartum hemorrhage should be admitted to the hospital for careful assessment, this is not of great practical importance. Of greater potential danger is the patient who has abruptio placenta with concealed hemorrhage who may have little pain from the distending uterus and may delay calling her physician until vaginal bleeding, incipient shock and peritoneal pain occurs. The delay of even an hour in such a patient's admission to the hospital may lead to maternal exodus from shock, hypofibrinogenemia, postpartum hemorrhage or, at a later stage, from bilateral renal cortical necrosis. Early and

adequate treatment is essential if maternal and fetal mortality are to be decreased from abruptio placenta.

3. Precipitate Delivery. Precipitate delivery at home and unattended is liable to occur and the dangers of this to the mother and baby are obvious.

4. Oxytocic Drugs. Dangers of stimulation of uterine contractions by oxytocic drugs in a patient already in active labor must be appreciated. Many of these patients make no complaint of pain, although cervical dilatation is well advanced. The attending staff must be cognizant of this fact, otherwise they may, without palpating the abdomen, prescribe pitocin to stimulate contractions. This may result in precipitate delivery with injury to mother and child or even rupture of the uterus, especially where an unstable vertex becomes an abnormal presentation.

SUGGESTED MANAGEMENT

1. All postneurectomy patients should be aware of the likelihood of an altered labor pattern. Special emphasis should be placed upon the insidious onset and the diminution of conscious first stage labor pain. They should be instructed to call the obstetrician if in doubt and to go immediately to the hospital in the event of ruptured membranes or bloody show. We should also mention that the surgeon who performs the operation of presacral neurectomy should impress upon his patient the possible changes in the normal pattern of labor which may occur in order that she may pass this information on to her attending obstetrician.

2. Obstetrical nurses and resident staff of the hospital should be fully aware as to what to expect in these patients. On admission the patient should be examined abdominally and vaginally. The abdomen should be palpated every 15 min. and contractions charted once it has been decided that the patient is in labor. This should be done every hour even when the patient is not in apparent labor unless the cervix is thick and closed and the presenting part is free.

3. An important feature of prenatal care is weekly vaginal examinations in the last four weeks of pregnancy, unless of course this is contraindicated by obstetric conditions such as bleeding.

4. The possibility of surgical induction should be considered in a patient who has a history of previous rapid labors and in whom the examination reveals an engaged vertex with ripe cervix, and this fact should be discussed with the patient. The final decision regarding surgical induction should be that of the patient.

5. Unless contraindicated by prematurity we believe that sedation should be given at about 6-cm. cervical dilation in the primigravida and at 8-cm. dilation in multigravida, even if no excessive discomfort is present at this stage of labor. This reduces terminal first stage discomfort and second stage pain as well as allaying anxiety in the mind of the patient. It also aids in the administration of local or general anesthesia for the delivery of the child.

6. General or regional anesthesia may be used for delivery. It is our practice to use saddle-block anesthesia unless contraindicated, and we find it most satisfactory for episiotomy and forceps delivery. Pudendal block is quite adequate for the relief of pain of perineal distention.

7. We would suggest that adequate prenatal summaries should go to the hospital at least two weeks prior to the expected date of delivery of each patient. This summary should be attached to the patient's hospital chart for the benefit of the house officers, nurses, and the attending physician.

SUMMARY

1. Pertinent points in anatomy are reviewed, in particular those having to do with variations of the "presacral nerve."

2. The five principal pathways of labor pain are described.

3. Ten case observations are reported in eight patients who had previous presacral neurectomy.

4. Conclusions drawn from these cases are correlated with those of other authors.

5. There is discussion of typical and atypical postneurectomy labor patterns and their explanations.

6. Important practical aspects of these cases are emphasized.

7. An outline of suggested management is given.

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SUBFASCIAL TENSION HEMATOMA AND IMPENDING ISCHEMIA RESULTING FROM CONTUSION OF THE FOREARM: CASE REPORT

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Seldom does the clinician have an opportunity to observe and record the primitive dichotomy of human wounding—insult and reaction—such as that which is produced by a single contusive blow. The case to be reported is one of these exceptions. It also illustrates that a mechanical injury is a continuous event beginning from impact and resulting in the fully developed reactive lesion. Moreover, these events lend perspective to biomechanics involved in the evolution of the complex called Volkmann's ischemic paralysis and/or contracture.

CASE REPORT

A batter missed a pitched baseball and struck instead the upper volar surface of the right forearm of a 33-year-old catcher at the end of his swing. The injury was received at 7:30 in the evening. There was moderate discomfort to begin with. Within several hours the pain became progressively more severe and spread to the hand and fingers, and 4½ hr. after injury he was admitted to the hospital. Examination showed the volar surface of the affected forearm to be swollen, extremely tender at the junction of the middle and upper one-thirds, tense, and the superficial veins prominent and engorged (fig. 1). However, there were no external signs of bruising. The radial pulse was diminished to the point of absence. The fingers were held in the slightly flexed position and were painful to passive attempts to straighten them. There was paresthesia in the hand and fingers which could be roughly mapped to involve the distribution of the median and, to a lesser extent, the ulnar nerves. X-ray examination showed no evidences of skeletal injury but did outline a subfascial, more or less localized, swelling of the soft parts. The clinical diagnosis was postimpactive subfascial tension hematoma with an impending Volkmann's type of ischemia. Because the man's pain was becoming increasingly more unbearable, operative exploration and subfascial decompression was done under sodium Pentothal anesthesia 5½ hr. after his injury.

The deep (ensheathing or vaginal) fascia of the front of the forearm was exposed through a 4-in. incision (fig. 2 *A*). There was no evidence of injury in the subcutaneous tissues. The fascia appeared to be darker than normal in color, which was assumed to be due to free blood directly beneath it. The fascia was then incised (fig. 2 *B*) but the expected free blood was not found. Instead, the superficial muscle bellies were moderately swollen and mildly infiltrated with blood. These mucles bulged out of the fascial wound as though something underneath was forcing them out. Digital exploration approximately at the apex of the antecubital fossa revealed the latter to contain a well-defined clotted and liquid blood which was carefully evacuated (fig. 2 *C*). Some of the deeper muscle fibers were damaged and the muscle bellies were grossly infiltrated with blood. Several bleeding points were ligated. There was no injury to nerves.

Only the skin was closed loosely without drainage. The patient was completely relieved of pain and he was discharged from the hospital 11 hr. after admission. The skin healed *per primum* within a week. There were no further complaints or complications.

DYNAMICS OF THE INJURY

The essential character of the force was pressure exerted by the bat for a very brief period of time. Also, the acceleration/deceleration of the arm was moderated by the natural recoil instinctively activated by the patient at the moment of contact. This more or less "roll-with-the-punch" reaction has clinical implications with regard to manifestations of injury of mechanical origin. This probably explains the lack of signs of localized injury to the skin and subcutaneous tissues. The perfect corollary to this situation may be found in the discrepancies frequently noted in injuries to the head, chest, and abdomen. Not infrequently internal injuries are received without external evidences of injury, *i.e.*, cerebral damage without scalp or skull damage. The same is to be noted with regard to injuries of the chest and abdomen. In other words, there can be no mechanical injury unless there has been an adequate absorption of energy.

In the case under discussion, the energy ab-

FIG. 1. Appearance of patient's right forearm 4½ hr. after receiving a single blunt blow to the upper volar surface from a wielded bat. The localized swelling and distended veins are notable. The fingers were slightly flexed and painful to passive stretching.

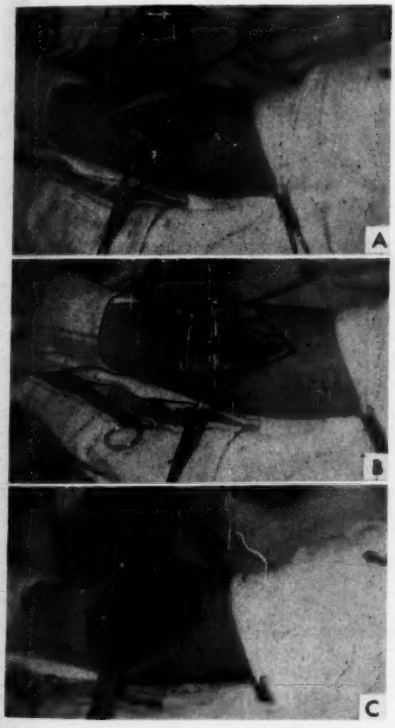

FIG. 2. *A, top:* Appearance of volar fascia 5½ hr. after injury; *i.e.*, showing dusky hue and tension; *B, center:* Swollen dusky appearance of the muscles immediately below the fascia after the latter had been incised. There was no free flow of blood at this time; *C, bottom:* Good sized hematoma and free blood evacuated from the deepest layer of muscles by blunt dissection at the apex of the antecubital space.

sorption took place almost wholly by the deepest layer of muscles which had been jammed against the forearm bones, *i.e.*, the area of most rapid acceleration of the tissues. However, the brevity of the force's application and its relatively wide distribution prevented excessive crushing of the muscles under impact. Instead, the most aggravating factors following injury were derived from the steadily increasing subfascial tension resulting from hemorrhage, swelling and edema of these muscles. This could only have happened either within a closed space such as normally present in the subfascial area of the forearm or wherever there is an ensheathing or vaginal fascia.

PATHOLOGIC PHYSIOLOGY

Figure 3 indicates the dual role of the deep or vaginal fascia. In the first place it serves to separate the superficial veins from the deeper arteries and veins (also muscles and nerves). Secondly, it encloses all of the deeper structures within a closed space—most notably at the elbow and forearm. The deep fascia yields very little to increasing hydraulic pressure beneath it. By the same token, when subfascial tension does exist, the superficial veins are particularly vulnerable to external pressure which is brought to bear, possibly, by bandages, splints, or plaster encasements in the treatment of fractures about the elbow joint.

However, in this case, it was clinically evident that both epifascial (see distended veins on the forearm shown in fig. 1) and subfascial vascular embarassment was present, without the incriminating aid of externally applied pressure from bandages, splints, or plaster encasement. This statement does not mean to subordinate the

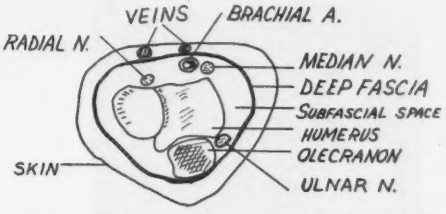

FIG. 3. Drawing of cross section through the right elbow joint looking proximally. The dual role of the deep or vaginal fascia is notable; *i.e.*, separating the superficial veins from the enclosed space beneath the fascia, and its envestment of all subfascial structures.

possible role played by the latter in the production of ischemic states. Such an interpretation would tend toward therapeutic carelessness and/or frustration with regard to prophylaxis when ischemia impends or threatens. I wish to stress this dual primary effect of the deep fascia which should tend to make clinicians even more alert to the devastating possibilities regarding both venous and arterial embarassments not infrequently noted in injuries of the elbow and forearm.

The primary role played by the deep fascia in the production of Volkmann's ischemia is also reflected in its thickened and constrictive condition which is noted during operative rehabilitation of irreversible contractures; the incision of which seems to relieve at least some of the cohesive constriction of the muscles, nerves and blood vessels—even before individual release of adhesions.

CLINICAL COMMENT

The inciting injury in most cases of Volkmann's ischemia of the forearm is the extension type of supracondylar fracture of the elbow in children. Some of the causes other than fracture that have been stated to be of damage to the brachial or axillary artery are, embolus or rupture, subfascial hematoma from a ruptured artery or hemophilia, exposure to cold, and, primary or secondary so-called reflex segmental arterial spasm. Undoubtedly, other known etiologic factors such as external pressures also play a part, in addition to some unknown ones. Most cases observed clinically have been complicated ones. This patient's report comprises a relatively uncomplicated type of impending Volkmann's ischemia which was the result of a single primitive blow not attended by fracture. Complete cure was achieved by simple subfascial decompression, *i.e.*, incision of the fascia and evacuation of the tension hematoma about the deepest muscular layer of the forearm.

Fortunately, full blown cases of Volkmann's ischemic contracture are relatively uncommon. Threatening or impending ischemia however, is seen more frequently. I have seen a number of these cases complicating fractures of all kinds

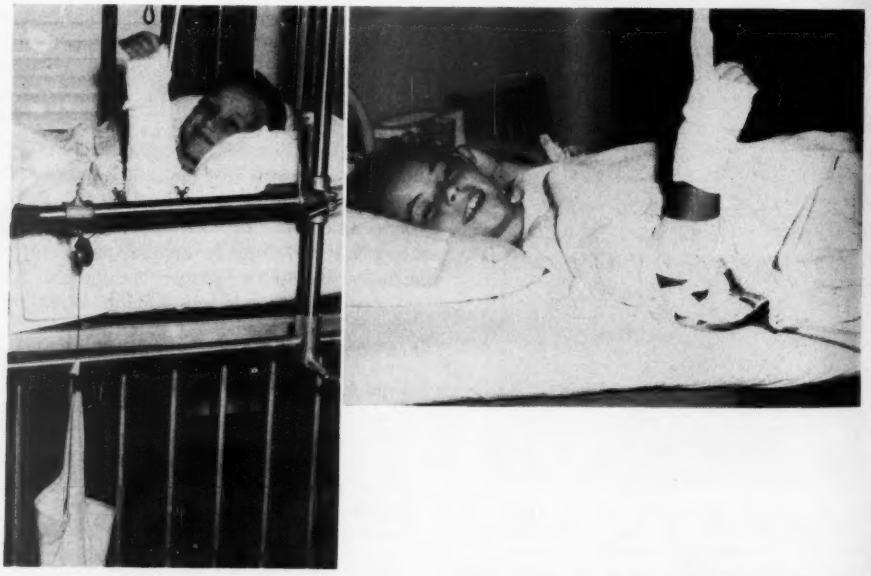

FIG. 4. Impending Volkmann's ischemia is not a rare occurrence; these two children—brother and sister—were treated for severe complicating extension supracondylar fractures within a period of two weeks. The boy at the left was operated upon and showed a classical segmental reflex arterial spasm of the brachial artery 24 hr. after injury. He recovered after a period of traction and casting. The girl, on the other hand, got well after skeletal traction and injection of procaine hydrochloride in the vicinity of the median nerve.

about the lower arm (once in a fracture of the clavicle), elbow, forearm and even occasionally the wrist. When ischemia threatens, the simplest measure which can be instituted positively is subfascial decompression. This can be done by surgical exposure and single or multiple interrupted fascial incisions or, as this writer prefers, by careful multiple punctate subfascial stab incisions with a pointed knife blade. In a series of about 20 such operations, one case was further complicated by infection and somewhat persistent swelling, but, as in the others, the ultimate result was good.

It must be emphasized that, concurrently with subfascial decompression, all other precautionary steps should be taken; *i.e.*, release of bandages or splints (plaster or otherwise) that are too tight, decrease of the angle of flexion of a reduced elbow fracture, and, properly applied traction—preferably overhead and skeletal (fig. 4). The latter may be adopted as an excellent preventive treatment, in the first place, when the doctor is confronted by an elbow fracture which has lacerated the musculature about the elbow to the point of endangering the neurovascular bundle (brachial artery and median nerve).

When these simple primary measures have failed to improve the situation (especially when complicated by fracture) the question of exploration of the brachial artery in the antecubital fossa must be carefully weighed and preformed as indicated. My first observation of so-called segmental arterial brachial spasm occurred in a severe *compound* extension type of supracondylar fracture in a child. Tearing of the fascia and other soft tissues had obviously decompressed spontaneously the subfascial space. This was not, then, a factor in the impending ischemia in this particular patient. The artery was explored and was found to be extremely constricted. Dramatic relief of spasm was obtained from procaine injection (1 per cent) into the undamaged median nerve.

SUMMARY AND CONCLUSIONS

A case of impending Volkmann's ischemia of the forearm due to a single blunt blow, and not complicated by fracture, is reported. The dramatic response to surgical subfascial decompression suggests that this simple method of treatment should be given priority in the more complicated cases of fractures about the elbow in which ischemia is a dominant factor. The primary role of the deep fascia in governing the development and evolution of this type of ischemia should tend to subordinate all other etiologic mechanisms.

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MUSCLE RELAXANTS IN MANAGEMENT OF ORTHOPEDIC BACK INJURY

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Orthopedic disorders of the back are largely a result of civilization. Accidents, particularly those involving vehicles in motion, account for many such disabilities. Overexertion of untrained or undisciplined muscles can lead to the painful disablement so well known to the enthusiastic week-end gardener. Poor posture contributes to about 60 per cent of back-pain complaints seen in orthopedic practice.

Conservative management of such disorders calls for repair of the injury and a balanced physiotherapeutic program, supplemented by analgesics and superficial or deep heat. Under such a regimen, most patients sooner or later resume normal activity with little if any permanent damage to the ligamentous tissues.

To the patient worried over his job or earning capacity, the phrase "sooner or later" is disheartening. To the orthopedic surgeon, prolonged rehabilitation rashly encourages invalidism. With certain exceptions obvious to the experienced practitioner, early introduction of the orthopedic patient into a gradually accelerating program of physical therapy promotes more rapid restoration of normal function.

Past efforts to accelerate rehabilitation of patients with orthopedic injury have lacked signal success for at least three reasons.

Obviously, pain will limit patient efforts. Regardless of cooperativeness, it is hard for most patients to believe that exercise of the injured part against the inhibition of pain can prove beneficial. Analgesics help control pain, but in themselves do not aid in restoring normal function to the injured area.

Muscle spasm not only slows response to exercise, but also triggers the pain reflex, and so retards rehabilitation. Muscle tension, restricted motion, and point tenderness can all be associated with this condition.

A third and perhaps less well-recognized factor in rehabilitation of orthopedic patients is the presence of emotional trauma. The shock of accident, and the mental response that follows, establishes in many patients a state of anxiety and tension. The relationship between anxiety, muscle spasm, and pain is accepted by most clinicians. Concern over inability to resume normal activities can produce or prolong patient anxiety, and hence muscle tension and pain.

An adequate modern therapeutic regimen for rapid patient rehabilitation should thus provide: (1) care and repair of the injury, (2) muscle relaxation, (3) assuagement of anxiety, and (4) relief from pain. Agents to achieve these last three goals should be administered in quantity sufficient to enable restoration of normal function as rapidly as is consistent with the severity of the orthopedic disorder.

If spasm is present, at least partial relief from pain follows administration of muscle relaxant agents to orthopedic patients. Many drugs and drug combinations will control muscle spasm and relax skeletal muscle. Agents used earlier had certain disadvantages; curare, for example, presented the difficulty of achieving muscle relaxation without producing flaccid paralysis. Modern muscle relaxants in use include zoxazolamine (Flexin), methocarbamol (Robaxin), mephenesin, and such mephenesin derivatives as meprobamate (Equanil).

Mephenesin administered alone or with analgesics and sedatives has attained some acceptance.[5] However, mephenesin is reported to be relatively short-acting and poorly absorbed orally; hence, therapeutic doses are large. Smith, and associates,[6] have noted that relief of muscle stiffness in rheumatic disease requires 0.5 gm. of mephenesin for each 46 lb. of body weight as an initial dose, followed by identical doses every three hours throughout the day.

Zoxazolamine is reported to produce satisfactory muscle relaxation.[7] Vazuka[8] has compared methocarbamol, zoxazolamine and meprobamate in their effect on human muscle hyperactivity. He concludes that meprobamate is more effective than either zoxazolamine or methocarbamol as a muscle relaxant.

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None of the preceding drugs except meprobamate were noted to possess anti-anxiety properties. The tranquilizing action of meprobamate is well attested by clinical investigators.[3, 4]

Meprobamate at present appears unique among drugs available to the orthopedic surgeon. Its interneuronal blocking action produces relaxation of skeletal muscles without impairment of muscle function. It also acts on tissues in the subcortical area resulting in assuagement of anxiety and calming of the patient who is made chronically irritable by pain.[1] Side effects other than transient drowsiness are infrequent.[2]

PLAN OF STUDY

There were 129 patients seen in private and hospital orthopedic practice who were treated with meprobamate. All displayed typical symptoms of injury to the muscular or ligamentous structures of the cervical, dorsal, or lumbosacral spine. Disorders involving bone damage were omitted from the study. Ages ranged from 28 to 60 years, with a median age of about 45 years.

Most patients began meprobamate therapy within two days of diagnosis, and in accident cases from two to seven days after the injury. Duration of therapy varied with the severity of the orthopedic disorder, and ranged from 1 to 31 days. Patients with severe back injury requiring hospitalization received meprobamate not only during their hospital stay, but also for approximately two weeks thereafter. Generally, the drug was withdrawn at the patient's last office visit.

A standard dose of meprobamate (400 mg., four times daily) was administered regardless of age, weight or severity of the disorder.

Supplemental therapy usually included rest, regular application of local heat, and physical therapy. Among the 43 patients hospitalized immediately after their injury, meperidine hydrochloride was available for relief of pain while bedfast. No other analgesics were administered at any time during the study, except for salicylates in the office to an occasional patient with obviously severe pain. No muscle relaxant or anti-anxiety agents were given other than meprobamate.

CERVICAL INJURY

So-called whiplash injury to the cervical spine is a misleading but nonetheless descriptive term. Results of such injury can be serious, and can include fractures of the upper vertebras, as well as their facets, and pedicles. Prolonged immobilization in casts may be required.

Less serious cervical injury can involve ligaments, capsules of the cervical facets and inner osseous ligaments. Under roentgen examination, damage to these three areas may be undetectable. Even so, such an injury does cause great pain and discomfort. Muscle spasm aggravates this pain. The psychic trauma is as great as actual damage to ligamentous or neuromuscular structures.

Patients with cervical spine injury can best be treated with head traction and hospitalization for several days to several weeks. When ambulatory, a Thomas-type collar support may speed recovery, as will physical therapy.

REPORT OF CASES

Meprobamate was administered with excellent results to 36 patients having cervical spine injury. In our opinion, patients had less pain than did prior patients with similar injuries treated with other drugs. Rehabilitation was accelerated and patients returned to their gainful occupations sooner than if meprobamate had not been given.

Two surgeons with severe ligamentous injuries were included in this group. Under meprobamate therapy, these physicians lost no time from their practices. Both continued to work while recovering from their injuries. Later x-ray examination showed no appreciable residual damage.

TRAUMATIC BACK INJURY

Mid and low back injuries are common, particularly those associated with automobile accidents. Such injuries can be disabling and frequently require hospitalization. Severe pain occurs, as does muscle spasm, restriction of motion and often point tenderness about the various ligaments and paravertebral muscles.

Relief of muscle spasm is the first step in treatment. Restrictive therapy usually is required: a fracture board, a felt mattress, and leg or pelvic traction for five days to three weeks.

Throughout this prolonged period, physical therapy aids muscle relaxation and muscle group coordination Superficial or deep heat helps abused tissues to heal and relaxes individual muscle groups. Coordinated use of these muscles is extremely important to restore proper posture. Where pain is present, exercises must be carried out judiciously.

After release from the hospital, patients should sleep on a hard mattress. During the day, a corset or brace (the latter in severe injury) is needed for support. With muscles coordinated and pain relieved, healing can progress more quickly and the degree of permanent disability can thereby be reduced.

REPORT OF CASES

There were 26 patients with injury to the dorsal or lumbosacral spine who were treated with meprobamate. The drug appeared to render more tolerable the long hospital stay often required with such an injury. Meprobamate, we found, assisted the patient in acceptance of his disabled condition and contributed to his comfort. Interestingly enough, among patients covered by Workman's Compensation Laws, the drug lessened unwillingness to accept rehabilitation.

Once physiotherapy began, meprobamate was noted to relax the muscles and to facilitate muscle group coordination. When exercises were prescribed, the drug proved decidedly valuable. With meprobamate, the patient relaxed emotionally and physically, and exerted a greater effort toward individual adjustment.

The average patient with low or mid back injury spent about six days in the hospital, a shorter time than was possible prior to introduction of meprobamate. The drug was continued for five to seven days after discharge as healing progressed.

Where the orthopedic condition was of long standing, treatment was prolonged. Extended treatment also was required where marked contracture of the lumbosacral fascia had permanently tilted the pelvis. In this last condition, limb traction was most often undertaken to elongate the paravertebral lumbar muscles and the lumbosacral fascial aponeurosis.

LOW BACK PAIN

Low back pain frequently results from overexertion. The instructive exercises and muscle-building programs of childhood and young adulthood fully develop the body musculature, however, the pressures of our society tend to preclude regular exercise. Thus, most Americans in later life enter a general physical decline. Muscular strain from unaccustomed gardening is sufficiently familiar to be called a stereotype reaction. On Sundays, the average gardener (who has waited all week to care for his small plot) is permitted a few hours of heavy work. Typically, he works violently and without good muscular coordination. And typically, low back pain develops from excess strain on the fascial structures. While the injury heals, the patient often cannot follow his customary occupation.

Patients with low back pain are usually ambulatory. Treatment consists generally of a corset, local heat, and muscle relaxants to encourage healing and to promote comfort.

REPORT OF CASES

Meprobamate was given to 25 patients receiving treatment for low back pain. Duration of therapy ranged from one to seven days. Results were excellent in all cases. In our opinion, meprobamate reduced time lost from work.

POSTURAL BACKACHE

Postural backache is often seen today, even among individuals with excellent musculature. Bad posture is aggravated by high heels on the shoes of women, and by obesity and pendulous abdomens in both sexes. Poor chair posture may produce backache. Round-back deformities of childhood, regardless of etiology, are accentuated in later life and can cause backache. Such postural backaches pose problems in rehabilitation.

In treatment, relief of pain comes first. Sufficient rest on a proper bed will often achieve this. Suitable postural exercises assist, as does a surgical support in severe backache. Proper shoes and the proper gait also aid in correcting poor posture.

Frequently the preceding therapeutic regimen proves inadequate. The patient finds it hard to believe that bad posture alone could bring about his acute discomfort. More specifically, the corrective exercises can cause pain when first attempted, and most patients lack sufficient incentive to override the initial discomfort.

REPORT OF CASES

There were 42 ambulatory patients with postural back pain who received meprobamate as an adjunct to therapy where previous treatment had failed. In all cases, earlier therapy had proved inadequate because of patient unwillingness to follow a strict rehabilitation program that included corrective exercises.

The drug satisfactorily relieved muscle tension. Patients then found corrective exercises easier to perform with less pain. They became more willing to attend closely to the therapeutic regimen. Normally, drug treatment for five days proved sufficient to permit the patient to establish new

postural habits without undue pain. After this time, pain associated with poor posture (and the exercises necessary to correct the condition) diminished to tolerable levels, and drug therapy was suspended.

SIDE EFFECTS

Transient drowsiness was seen occasionally in our series, and was considered an advantage rather than a side effect. Two patients with traumatic back injury developed urticaria. The drug was withdrawn and the urticaria promptly subsided. No other side effects or evidence of intolerance appeared.

SUMMARY

Meprobamate, as an adjunct to conventional therapy, was administered (400 mg. four times daily) with excellent results to 129 patients with orthopedic disorders of the back. Analgesics were restricted. Duration of therapy ranged from 1 to 30 days. The drug functioned not only to relax abused muscular and ligamentous structures, but also to reduce patient anxiety over his disability. Response to physical therapy was enhanced, making possible an accelerated rehabilitation program. Patients most often returned to their occupations in less time than would have been possible under conventional therapy alone. Side effects were infrequent.

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PARATHYROID ADENOMA: REPORT OF A CASE*

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Since the first instance of a parathyroid adenoma operation reported in 1925 by Mandl,[12] approximately 600 such cases have been recorded in the literature.[8–13]

DEFINITION

Hyperparathyroidism is characterized by the presence of excessive amounts of circulating parathyroid hormone in the blood stream. It may be caused by an adenoma of a parathyroid gland, a carcinoma,[17–19] or primary hypertrophy and hyperplasia of the parathyroids. As stated by Dr. H. L. Jaffe, and quoted by Albright and Reifenstein,[1] it may be secondary to a chronic nephritis with concomitant hyperplasia of the parathyroid glands.

PHYSIO-PATHOLOGY

The presence of an excessive amount of parathyroid hormone in the circulating blood causes an increase in the level of serum calcium and decrease of inorganic phosphate. According to Albright and Reifenstein,[1] the parathyroia hormone acts on the electrolyte equilibrium of the body fluids and the bone changes are secondary to the chemical changes. The phosphate ion is believed to be affected by the parathyroid hormone in such a way that it is more easily excreted by the kidney. This produces a fall in the serum phosphorus level. Re-absorption of calcium and phosphate from bone occurs, thus increasing the calcium level and maintaining the equilibrium constant. The normal level of serum calcium of 10.0 to 12. mg. per 100 cc. is increased and the serum inorganic phosphate level falls below the normal of 2.0 to 4.0 mg. per 100 cc.

With mobilization of calcium in the blood stream, demineralization of bones occurs with consequent osteoporosis. In severe forms the bones become decalcified and fibrosed and present the clinical picture of osteitis fibrosa cystica. Increased urinary excretion of calcium and phosphorus may result in the formation of renal calculi.

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SIGNS AND SYMPTOMS

The most common findings are renal calculi and the bone changes are seen on x-ray. Bone enlargement, bone pain, or a pathologic fracture may be the first symptom. There may be gastrointestinal symptoms such as nausea, vomiting, or constipation and urinary symptoms such as polyuria and polydipsia. Joint pain and itching may be the only manifestations of the condition. In some patients demineralization of vertebras may be sufficient to cause shortening and deformity of the spine. General malaise and easy fatigability can be prominent features of the disease.[2,4,10,11,14,16]

In our patient, as in most of the cases reported, the outstanding feature was kidney colic associated with multiple calculi and general malaise.

DIAGNOSIS

In hyperparathyroidism there is hypercalcemia, with the value for serum calcium elevated above the normal of 12 mg. per 100 cc. Multiple determinations are necessary to establish its constancy. There is hypophosphatemia; the serum inorganic phosphate is below 2 mg. per 100 cc. The serum phosphatase activity is increased depending upon the severity of the bone lesions. The degree of this activity ranges between 8 and 35 Bodansky units (normal 2 units).

The calcium and phosphorus balance studies are of value. If the patient is placed for three days on a low calcium diet, the loss of calcium in the urine is much greater than normal. The Sulkowitch test determines the level of calcium excreted in the urine. X-ray examination may reveal the presence of osteoporosis, osteitis fibrosa cystica, and pathologic fractures as well as renal calculi.

The possibility of renal rickets, metastatic carcinoma, multiple myeloma, hypervitaminosis D, senile osteoporosis, idiopathic steatorrhea, and Paget's disease should be excluded.[3–7]

TREATMENT

The treatment of parathyroid adenoma is surgical removal. When a diagnosis of hyperparathyroidism has been established and surgical exploration fails to reveal an adenoma, it has been recommended by some authors that all but one of the parathyroid glands should be removed. This clinical entity can be caused by excessive secretion of parathyroid hormone by normal or hyperplastic parathyroid glands.[5, 6, 9, 15, 18]

CASE REPORT

N. H. H., a 53-year-old white woman was admitted to the hospital March 7, 1955, with the chief complaints of blood in the urine, of three to four weeks' duration, together with mild abdominal pain. There was no recent history of severe kidney colic. By intravenous pyelogram a calculus was identified in the right ureter.

The past history revealed that a kidney stone had been passed spontaneously five years previously, at the time of hospitalization for a hemorrhoidectomy.

Retrograde pyelography demonstrated multiple calculi in the left kidney and one small calculus in the inferior pole of the right kidney. There were no opaque calculi demonstrated along the course of either ureter.

LABORATORY FINDINGS

Urinalysis showed occasional red blood cell, white blood cell, and calcium oxalate crystals—serum calcium, 12 mg. per 100 cc.; serum phosphorus, 3 mg. per 100 cc.; and alkaline phosphotase, 4/unit. Roentgenograms of the skull were negative. The Sulkowitch urine test was normal, and when repeated the next day, it showed increased calcium. Subsequent studies showed a serum calcium of 12.4 mg. and a serum phosphorus of 3.5 mg. Repeated Sulkowitch tests showed a progressive increase of calcium excretion.

On March 21, 1955, under general endotracheal anesthesia, an enlarged parathyroid was excised from the lower pole of the right lobe of the thyroid. The superior pole was explored and a small parathyroid gland was identified and left in place. Two nodules were removed from the left lower thyroid pole which possibly could represent parathyroid glands. No parathyroid gland was found on the left superior pole. The next day, the patient developed edema of the larynx, necessitating a tracheotomy. The tracheotomy tube was removed on the sixth postoperative day. Subsequent recovery was without event.

FIG. 1

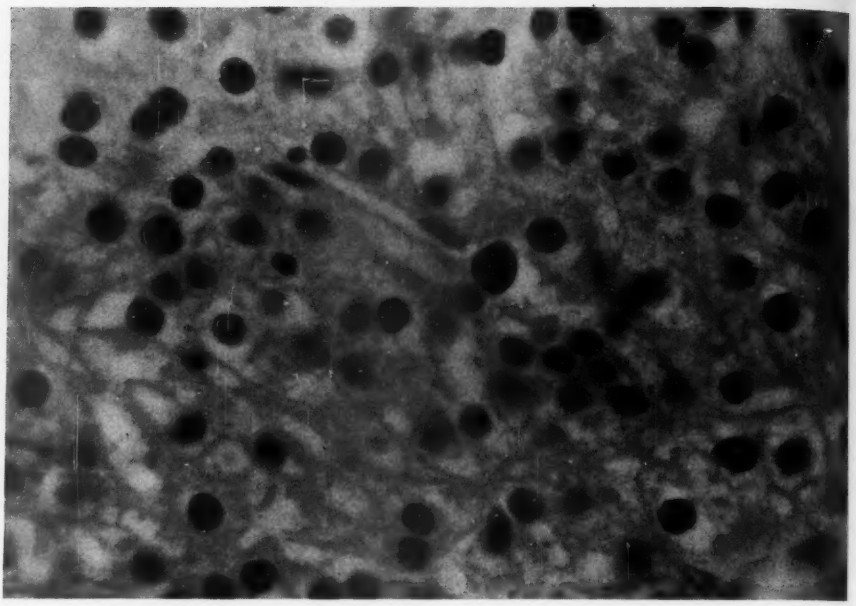

FIG. 2

PATHOLOGY REPORT

Microscopic (figs. 1 and 2): the adenoma was surrounded by a thin fibrous capsule. The major portion of the gland consisted of a monotony of small, uniformly sized mononuclear "chief" cells, having round, compact nuclei and finely granular or vacuolated cytoplasm with distinct cytoplasmic membranes. In some places the cells were grouped together in alveolar formations. In other portions of the gland there was more of an adenomatous appearance where the formation of glandular acini constituted the predominant picture. The oxyphil and wasserhelle cells were rarely encountered. Some portions of the adenoma appeared to be surrounded by a peripheral rim of smaller, more compactly arranged cells which gave the impression that the adenoma had formed within the interior of the gland and was compressing a peripheral rim of normal parathyroid cells.

On March 29, 1955, the day she was discharged from the hospital, the patient's serum calcium was 9.7 mg. per 100 cc. and her serum phosphorus was 3.8 mg. per 100 cc. She was discharged, completely recovered. When seen in the follow-up clinic in May 1957, her general condition was excellent She had no complaints.

SUMMARY

A brief review of the physio-pathology and treatment of parathyroid adenoma, with a case report, is presented.

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Editorial

THE OPERATING ROOM AS A SOURCE OF WOUND INFECTION

A great many people are justly alarmed at the epidemic of infection in supposedly clean wounds, which is sweeping over the country. There is much speculation as to the cause of this. Has the more or less indiscriminate use of antibiotics increased the resistance and hardihood of some organisms? Has the use of antibiotics caused a laxity in our surgical technique? Has the resistance of patients been lowered by too much antibiotic protection?

The organism involved is almost always *Staphylococcus aureus*. The work of Hart and Gardner has pretty conclusively shown that the main source of this organism is from the nasopharynges of the members of the operating team, the patient, and visitors in the operating room. They showed that Petri dishes exposed in the operating room almost aways grew cultures of *Staphylococcus aureus*. The same organism was present in their infected wounds. The number of infections on the Petri dishes was directly proportional to the number of people in the operating room, and likewise the number of wound infections was in the same proportion. They were able to cut down the number of Petri dish cultures and also the number of infected wounds by the use of ultraviolet lights, which they demonstrated would kill the bacteria. Since they have been operating under these lights, they have reduced their unexplained wound infections to almost zero.

In spite of the convincing work of Hart and Gardner, the profession as a whole has not become convinced of the practicability of using ultraviolet rays, possibly for two reasons. In the first place, the lights are expensive to install. In the second place, the eyes and the skin of the operating team must be protected from the effects of the rays in order to prevent burns. It has, therefore, been said that the method is both expensive and cumbersome. Those at Duke University do not find the method awkward, but even if it is somewhat cumbersome and expensive, the fact that it might reduce our operative infections to a minimum, may sooner or later make it necessary for all of us to use it. Almost any method that will eliminate the all too frequent trouble some infections in "clean" wounds is worthwhile adopting.

But aside from this, it can be easily shown that our operating room habits are not as meticulous as they should be. Hart and Gardner have shown that when an operating room lies "fallow" for a period of time, fewer infections are contracted in it. Operating rooms used alternately and not steadily have fewer infectious organisms than those which are used constantly. This shows that those which are constantly used are constantly being "seeded" with offending organisms. However, one not infrequently sees house officers, nurses, and orderlies going about operating rooms between operations with their masks down, thus unnecessarily adding to the bacterial flora circulating in the atmosphere of the room.

Furthermore, the masking of the operating room personnel is often not what it should be. Many house men wear their caps at a rakish angle, not entirely covering all of the head and forehead that the cap would cover if it were placed squarely on the head without having the edges turned up. A mask that covered the entire head and neck with only an opening for the eyes would certainly decrease the "fallout" of bacteria from exposed surfaces. Would it not be better also to have the part of the mask in front of the nose and mouth made of some impervious material to prevent its becoming moist, and thus allowing bacteria to penetrate it?

Some nurses have the habit of slipping their masks under their noses when they step out of the operating room and then back over their noses when they re-enter the operating room. Such a habit allows the breath of their nostrils to soil the outside of the mask and render it a source for disseminating organisms in the operating room. Personnel who cannot breathe adequately through a mask for hours on end have no business in an operating room.

Many house men are not particular about the wearing of their masks and do not change their masks often enough. Certainly masks which are not impervious should be changed frequently. Some of the worst offenders are anesthetists. One frequently has to ask anesthetists to keep

their noses as well as their mouths covered during the course of an operation.

Unnecessary talking, and especially laughing and guffawing, make the mask more penetrable to bacteria. Some surgeons wear two masks, but this is almost useless unless the rest of the operating room personnel are required to do the same thing.

One of the worst of all practices is failure to mask patients who are being operated upon under spinal or local anesthesia. Yet this is very commonly not done, and, during the operation they breath offending organisms into their own wounds.

Several studies have shown that *Staphylococcus aureus* is more frequently found in hospital personnel than in people who do not habitually work in hospitals. It is also more frequently found in "in-patients" than in "out-patients." Wound infections are fewer in summer than in winter, possibly because there are fewer organisms in the nasopharynges of hospital personnel during the warm season. For the same reason there are apparently fewer infections in the tropics than in temperate climates. One of the leading surgeons in Honolulu reports that he does not have wound infections there.

From the foregoing it is obvious that we need to make some drastic changes in order to eliminate unnecessary wound infections. In the meantime some surgeons have reduced the number of infections in their personal cases by placing 25,000 to 50,000 units of bacitracin solution in wounds before closing them.

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Book Review

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Operative Obstetrics. By R. GORDON DOUGLAS, AND WILLIAM B. STROMME. 735 pp., Appleton-Century-Crofts, Inc., New York, 1957.

This new textbook was conceived and written specifically as a companion to *Williams Obstetrics.* It does not and is not intended to replace the standard obstetric textbooks. Consequently, the considerations are brief.

The book does cover the entire field of operative obstetric management in the greatest detail. The author never assumes any feature is so obvious that the reader need not be informed of it. While this results in a wealth of detail, which may seem redundant to some, the usefulness of the book is broadened to include all levels of training and experience.

The conventional first section devoted to basic anatomy is followed by a chapter on preoperative care and operative management, including an outstanding discussion of the various techniques of incising the abdominal wall.

The clear and thorough presentation of the analgesia and anesthesia problem not only describes each method but compares the advantages and disadvantages of each in different situations. This remains a controversial area in obstetrics and this book reflects the experience of the New York Lying-In Hospital.

The chapter dealing with infant resuscitation and care of the newborn is up-to-date and evaluates present resuscitation techniques in a lucid manner.

Surgical complications of pregnancy such as abortion, hydatidiform mole, chorionepithelio and ectopic pregnancy are all thoroughly di cussed. Many "pointers" that represent fin of surgical technique and which are not o present in textbooks are given in these chapte

The most outstanding area of this book is th large portion devoted to operative vaginal de livery. The collection of excellent photograp devoted to this subject is most complete, feat ing large plates with explanatory diagrams. If t technique of operative delivery could be learn entirely through text and illustrations, this boo could approach that result.

Cesarean section is also described in equal de tail and clarity. The discussion of destructiv procedures is particularly valuable, since few ob stetricians today gain much experience in thi area, yet the need for the performance of embry otomy occasionally presents itself.

All conceivable surgical situations which may be associated with obstetric surgery includin appendectomy, genito-urinary problems, varicos ties, breast lesions, tracheotomy, and a host o others are also treated.

The sections dealing with postoperative car and complications maintain the standard of maxi mum coverage and great detail.

The entire book abounds with excellent illu trations, most of them photographs. Anything which could possibly be depicted pictorially i illustrated, often to the extent of near duplicatio For example, there are no less than 15 photograp devoted to appendectomy, 8 photographs to il lustrate paravertebral block, and approximately 115 photographs concerning vaginal forceps opera tions.

This textbook is very complete, extremely de tailed and beautifully illustrated.

EDMUND B. MIDDLETON, M. D.